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# CHEMICAL CHANGES OCCURRING IN THE BODY AS THE RESULT OF CERTAIN DISEASES

## I. THE EFFECTS OF DIARRHEA, VOMITING, DEHYDRATION AND OLIGURIA ON THE ACID-BASE BALANCE OF THE PLASMA OF INFANTS WITH MASTOIDITIS *

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Recently Peters, Bulger and others [1] have discussed from a broader point of view than usual the acid base equilibrium of the plasma of adults in health and in disease. For several years we have had a similar interest in the chemical changes taking place in the blood of sick infants and children. Because the normal blood of the infant differs somewhat from that of the adult, containing about 10 per cent less electrolyte and protein, and because rapid and serious changes of great magnitude are frequently encountered in disease, it was thought that our observations might be of some interest, both in emphasizing the significance of the chemical changes in certain diseases already thoroughly discussed by Peters and his associates, and in pointing out changes occurring as the result of diseases more peculiar to infancy and childhood.[2]

It is the purpose in this paper to present data secured from infants suffering from diarrhea, vomiting and dehydration, the result of infection in the middle ear and mastoid. The clinical picture in this group of cases was similar to that often described as "alimentary intoxication."

The method of collecting blood was the same as that described in a previous paper.[3] It should be emphasized, however, that stasis of the blood before collection, such as might be caused by the application of a tourniquet, was avoided by securing the blood samples either from

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1. Peters, J. P.; Bulger, H. A.; Eiseman, A. J., and Lee, C.: Total Acid-Base Equilibrium of Plasma in Health and Disease, J. Biol. Chem. **67**:141 (Jan.) 1926.

2. The chemical changes in the body occurring as a result of vomiting have already been discussed in a previous paper. Hartmann, A. F., and Smyth, F. S.: Chemical Changes in the Body Occurring as the Result of Vomiting, Am. J. Dis. Child. **32**:1 (July) 1926.

3. Hartmann, A. F., and Smyth, F. S.: Chemical Changes in the Body Occurring as the Result of Vomiting, Am. J. Dis. Child. **32**:1 (July) 1926.

the superior longitudinal sinus or from the external jugular vein. The chemical methods were also the same as those described previously,[3] with the addition of the determination of total base, either by the method of Stadie and Ross [4] or by a slight modification [5] of the method of Van Slyke,[6] of lactic acid at first by the method of Clausen,[7] and more recently by that of Friedemann, Cotonio and Shaffer,[8] and of the freezing point of serum by means of a thermocouple and galvanometer, as described by Buckman and Darrow.[9]

In estimating the base-binding value of protein, the formula of Van Slyke [10] was used, derived from the experimental data on the base-binding capacity of the serum protein of the Manchurian pony. In calculating the base bound by carbonic acid, we disregarded the $p_H$, and assumed that 1/21 of the carbon dioxide content was due to free carbonic acid. The error introduced in this way is insignificant.

In the construction of the charts, designed to show all of the chief individual acids whenever possible, we placed BCl on the base line, and on top of it the other acids in the order of their normal relative values; i.e., first bicarbonate, then protein, then phosphoric acid and finally lactic acid. Whenever the experimentally determined total base exceeded the sum of these acids, the difference was indicated in the charts as B-R. On top of the combined total acid value, we placed the entire nonprotein nitrogen content, calculated approximately in terms of osmotically equivalent tenth-normal electrolyte.[3] In this calculation, we considered the entire nonprotein nitrogen as urea nitrogen.

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4. Stadie, W. C., and Ross, E. C.: A Micro-Method for the Determination of Base in Blood and Serum and Other Biological Materials, J. Biol. Chem. **65**:735, 1925.

5. Instead of determining the sodium iodate gasometrically, we preferred to determine it iodometrically, on the suggestion of Prof. P. A. Shaffer, by titration with 0.005 normal sodium thiosulphate solution after the addition of an excess of potassium iodide and sulphuric acid. A 2 cc. aliquot, equivalent to 0.16 cc. serum, in this way usually liberates from 30 to 35 cc. 0.005 normal iodine solution, which renders this part of the determination extremely sensitive and makes unnecessary the use of the manometric gas apparatus. For the determination of the total base in this manner, we are indebted to Dr. Ludwig Schoenthal, and for the freezing point and several of the total base determinations by the method of Stadie and Ross, we are indebted to Dr. Dan C. Darrow.

6. Van Slyke, D. D.; Hiller, A., and Berthelsen, K. C.: A Gasometric Micro Method for Determination of Iodates and Sulfates, and Its Application to the Estimation of Total Base in Blood Serum, J. Biol. Chem. **74**:659 (Sept.) 1927.

7. Clausen, S. W. : A Method for the Determination of Small Amounts of Lactic Acid, J. Biol. Chem. **52**:263 (May) 1922.

8. Friedemann, T. E.; Cotonio, M., and Shaffer, P. A.: The Determination of Lactic Acid, J. Biol. Chem. **73**:335, 1927.

9. Buckman, T. F., and Darrow, D. C.: To be published.

10. Van Slyke, D. D.; Wu, H., and McLean, F. C.: Studies of Gas and Electrolyte Equilibria in the Blood, J. Biol. Chem. **56**:804, 1923.

Short clinical abstracts of each case, covering only those points which were felt to be of interest or importance, and the chemical data in table and chart form follow.

## REPORT OF CASES

CASE 1.—Curtis T., an infant, aged 7 months, with a history of poor feeding was admitted to the hospital on Oct. 4, 1924. One week before admission, he developed fever, vomiting and diarrhea. The temperature at the time of admission was 38 C. (100.4 F.). There were marked athrepsia and anhydremia. Death occurred on the fifth day, at which time the temperature had reached 40 C. (104 F.). There was persistent watery diarrhea, but little vomiting. For three days preceding death, there were signs of bronchopneumonia. Daily parenteral salt or dextrose solution was given. His weight on the day of admission was 5,830 Gm.; on the day of death, 6,300 Gm.

CASE 2.—Frank S., an infant, aged 5 months, who had never done well on supplementary feedings, about a week before admission to the hospital developed severe diarrhea. On admission, Oct. 9, 1924, he was found to be athreptic and anhydremic. His weight was 2,620 Gm. This patient lived fifteen days after entering the hospital, during which time he showed all symptoms of infection with severe vomiting and diarrhea and marked hydrolability. Parenteral fluid, such as Ringer's or dextrose solution, was given daily in large amounts. Until he died, when there was evidence of a terminal bronchopneumonia, the infection seemed to be entirely in the upper respiratory tract, particularly in the middle ears and mastoids. There was moderate leukocytosis. The urine contained albumin and casts.

CASE 3.—Minnie S., an infant, aged 2 months, had been well until about ten days previous to admission to the hospital on Oct. 30, 1924. At that time, she had developed diarrhea, vomiting and fever. When admitted she was in extremis; her color was a cyanotic gray; the eyes were sunken; the muscles flaccid; she did not cry and was almost pulseless. There was extreme anhydremia. After intravenous and subcutaneous injection of saline solution, there was slight temporary improvement. The infant lived five days, during which she suffered from marked vomiting, diarrhea and persistent fever. Daily parenteral injections of fluid were given. The pathologic observations at autopsy were multiple hemorrhagic erosions of the mucosa of the stomach, hemorrhage without ulceration in the colon and acute mesenteric lymphadenitis. The mastoids were not opened.

CASE 4.—Kenneth H., an infant, aged 1 month, was admitted to the hospital on Jan. 12, 1925, because of fever, vomiting and diarrhea. He lived seven days after admission, during which time he showed all the symptoms of infection, with severe diarrhea and hydrolability. This patient had bilateral otitis media and mastoiditis, and at times, there was marked watery diarrhea. The temperature averaged 39 C. (102.2 F.). On January 16, the day before death, vomiting became a prominent symptom for the first time.

CASE 5.—John G., an infant, aged 2 months, had had numerous convulsions since he was 2 weeks of age. On March 25, 1925, he was admitted to the hospital because of high fever. At the time of admission, he was athreptic and anhydremic, with a rectal temperature of 40 C. (104.0 F.). He was in the hospital one month, during which time he presented all the symptoms of infection, together with gastro-intestinal symptoms of vomiting and diarrhea, and great fluctuations in weight. Convulsions occurred frequently, most of which were one-sided. Purulent otis media persisted; both mastoid antrums were opened and pus was found in each. During the last two weeks of life, the temperature was

high, and vomiting, diarrhea and loss of weight were marked. Daily injections of parenteral fluid were given. Marked symptoms of acidosis preceded death, which occurred on April 22.

CASE 6.—Leo S., an infant, aged 8 weeks, one of twins, was breast fed for five weeks and then put on several poor types of artificial feeding. He failed to gain in weight, vomited considerably, and when he was 5 weeks old had had several convulsions; following these convulsions, he continued to have fever, which reached 103 F. three days before admission. He was a small, athreptic, moderately anhydremic infant and weighed 2,050 Gm. The temperature during the first day in the hospital averaged 41 C. (105.8 F.). Bilateral otitis media was present, and this was considered the cause of the fever. After the ear drums were incised, the temperature fell almost to normal on the third day, and diarrhea ceased. The temperature quickly began to rise again, and diarrhea became marked. On the sixth day after admission, a bilateral mastoid antrotomy was performed, without any effect on either the temperature or the diarrhea. On the twelfth day the infant died; the temperature had fallen from 41 C. (105.8 F). to 36 C. (96.8 F.) during the last twenty-four hours. Fluid, in the form of parenteral injections of saline and dextrose, was given daily in large amounts. Three blood transfusions were given. On the seventh day, the weight was 2,400 Gm., and on the day of death 2,080 Gm.—slightly more than on admission. On the day of death, there was marked air hunger; acetone was not detected on the breath. The blood was taken on the third day for study.

CASE 7.—Selma G., aged 3 years, with a history of premature birth and apparent intracranial hemorrhage, which resulted in a spastic paraplegia and rather marked mental deficiency, was admitted to the hospital on June 15, 1925; she became ill three days before admission, with fever, vomiting and diarrhea. These symptoms persisted until death, on June 18. The clinical picture showed a virulent bacillary dysentery. There was also marked gingivitis and secondary anemia.

CASE 8.—Mary G., a premature infant, aged 4 months, weighing 2,650 Gm., was admitted to the hospital on July 8, 1925. A previous history could not be obtained. Until death, on July 13, the infant showed all the symptoms of infection, together with severe vomiting and diarrhea. Bilateral otitis media was present, and at times anhydremia was marked. Air hunger was present almost constantly. There were signs of pneumonia just before death.

CASE 9.—Mildred R., an infant, aged 4 months, was admitted to the hospital on Aug. 16, 1925, with fever and marked desiccation. Four days after the onset of the illness, the patient had a severe watery diarrhea. Until death, five days later, moderately severe vomiting, profuse watery diarrhea and fever persisted. There was temporary improvement after the first administration of saline and dextrose.

CASE 10.—Tommie C., an infant, aged 4 months, was admitted to the hospital on July 20, 1925, because of diarrhea, vomiting and fever of about two weeks' duration. The patient lived five days after entering the hospital, during which time there persisted fever, which apparently resulted from otitis media, moderately severe vomiting and watery diarrhea. Clinical acidosis was always present, and at times desiccation was marked.

CASE 11.—Dorothy B., an infant, aged 11 months, was admitted to the hospital on Sept. 18, 1925, because of vomiting and diarrhea. She had never been fed well. Seven weeks before admission, she had developed an infection associated with fever, cough, purulent rhinitis, vomiting and diarrhea. When admitted, she was rachitic, athreptic, anhydremic and acutely ill. Fine scattered râles were present

in the chest; both ear drums were dull gray and lusterless, and when one was incised, the middle ear cavity was found dry. The signs in the chest continued. Vomiting was marked, and slight diarrhea was present. The breathing became hyperpneic, and the infant died on September 23. In addition to a transfusion, frequent parenteral injections of salt and dextrose solution were given.

CASE 12.—Robert U., aged 10 weeks, was admitted to the hospital on Dec. 26, 1925. The birth of this patient and the first two months of life were said to have been normal. Two weeks before admission, he developed a "cold," and during the two days prior to admission, fever, diarrhea and right-sided convulsions were present. Frequent clonic convulsions of the right arm and leg were noted, and at times there was a marked irregularity of breathing. The urine contained considerable albumin, a few casts and many pus cells. The chemical changes in the blood were unusual, especially the high initial chloride content. Pyuria, associated with slight fever, persisted, and on December 30, sodium bicarbonate, 2 Gm. every four hours, was given by mouth. During the next three days, the temperature was higher, and there were some vomiting and mild diarrhea. The respirations became more shallow, and frequently there were long apneic pauses. Convulsions, occasionally bilateral, became more frequent. An extremely high sodium bicarbonate content of the blood was then found. Administration of alkali was discontinued. Dextrose was administered intravenously and the child was forced to take water. By January 12, the blood had become normal chemically. On January 15, an organizing blood clot was removed from over the left motor cortex. After this operation, convulsions did not occur, and the infant seemed to have recovered.

CASE 13.—Louis B., an infant, one of twins, aged 2½ months, had been given sweetened condensed milk feedings since he was 1 week old. On July 14, 1926, two days before admission to the hospital, he became feverish and extremely irritable, and the following day pus was seen discharging from both ears. At the time of admission, he was dehydrated. The temperature was 39 C. (102.2 F.). Alternate periods of stupor and irritability were present. Between spells of crying, the breathing was distinctly of the air hunger type. At times there was marked cyanosis, particularly of the extremities. Purulent bilateral otitis media was present, and on the left side there was a sag of the posterior superior canal wall. On July 14, 400 cc. of Ringer's solution was given intraperitoneally. On the next day, blood was taken for study. Ringer's solution intraperitoneally, dextrose intravenously, and later citrated blood intravenously were given, and a bilateral antrotomy was performed. Pus was found in both mastoids. The next day, although the temperature averaged 39.5 C. (103.1 F.), he seemed somewhat better, but was still dehydrated. More Ringer's solution was given. He died on the following day, shortly after dextrose solution was injected into the sinus. While in the hospital he vomited only once, but during the last two days there was watery diarrhea.

At autopsy pathologic evidence of mastoiditis was found, and a smear made from the pus obtained from an undrained cell showed streptococci. The gastrointestinal tract was normal.

CASE 14.—Marguerite G., an infant, aged 9 weeks, for the first two weeks after birth had been fed on evaporated milk and for the next three weeks on sweetened condensed milk. Because of loss of weight, a sweet cow's milk dilution with added dextrimaltose was given, and on this feeding the patient gained slowly. On Sept. 1, 1926, the day before admission to the hospital, she suddenly became ill. In twelve hours, she had about twenty watery stools. Later, coincident with the development of fever, several convulsions occurred. On admission, she was

athreptic and anhydremic. The temperature was 39 C. (102.2 F.). Both ear drums were gray and dull, and when opened, a thin bloody discharge escaped. Ringer's solution was given intraperitoneally, but in less than twenty-four hours death occurred.

CASE 15.—Isabelle G., aged 3 weeks, was small at birth and did not gain in weight well. Stools were frequent and watery almost from birth. On Oct. 25, 1926, the patient was admitted to the hospital with slight fever and marked watery diarrhea. Rhinitis and otitis media were present. The nutrition was extremely poor, and moderate anhydremia existed. On October 29, when blood was taken for chemical study, extreme hyperpnea was present. Previous to this, the infant had received intraperitoneal injections of Ringer's solution. Following the administration of sodium bicarbonate, hyperpnea became less, and for a short while breathing was normal. Diarrhea and watery stools continued, and hyperpnea again recurred. Sodium bicarbonate was again administered intravenously, with decreasing hyperpnea. The infant was obviously growing worse. A postauricular incision was made because of the otitis media present, with the hope of establishing better drainage. The infant died on October 31, at which time signs of terminal bronchopneumonia were present.

CASE 16.—Bonnie S., an infant, aged 2 months, was breast fed for three weeks and then given several kinds of highly diluted feedings, which were frequently changed because of vomiting and diarrhea. One week before admission to the hospital on Aug. 5, 1927, she had not taken any food except barley water. For two weeks there had been a slight, irregular fever. On admission she was extremely athreptic. Her weight was 2,700 Gm. There was also moderate dehydration. The right ear drum was dull red, but not bulging. The left ear drum seemed normal. Lactic acid milk with Karo syrup was given. Ringer's solution was injected intraperitoneally, and a transfusion was performed. During the next five days, a rapid gain in weight of 570 Gm. occurred. A bilateral otitis media with fever, moderate vomiting and diarrhea and loss of weight then occurred. After incisions were made in the ears, the temperature became normal, and the patient gained in weight for a week or so. The ears continued to discharge pus, however, despite adenoidectomy and local treatment of the nose and ears. Frequent exacerbations of fever, vomiting, diarrhea and loss of weight occurred, and on September 22, a bilateral antrotomy was performed. Considerable thick pus with excessive granulation tissue and some necrosis of the bone was found. After the operation, the infant received a transfusion and was given Ringer's solution intraperitoneally. The next day marked air hunger was in evidence. More Ringer's solution was given. The temperature steadily rose, and the patient died on September 24. Shortly before death, blood was taken for study.

CASE 17.—Denise C., an infant, aged 2½ months, who had been born from three to four weeks prematurely, had for two months been breast fed, and then because of the illness of the mother, she had been weaned and given diluted sweet milk, to which dextrimaltose had been added. The patient did well on this feeding until she developed diarrhea, six days before admission to the hospital, on Oct. 18, 1927. The diarrhea, at first mild, became severe just before admission. There had been little vomiting and slight fever. She was dehydrated and much prostrated. Bilateral otitis media was present. Ringer's solution, intraperitoneally, and dextrose, intravenously, were given on October 19 and 20. Air hunger was frequently present; vomiting and watery diarrhea persisted. On October 20, a bilateral antrotomy was performed. On one side, an unusually large antrum was found, filled with thick yellow pus. A smaller antrum, with thinner pus, was present on the other side. After operation, a transfusion was performed. The next day,

more Ringer's and dextrose solutions were given. Air hunger became more marked, and the patient died. A half hour before death, blood was taken for chemical study.

CASE 18.—Salvatore R., an infant, aged 2 months, after being breast fed for a short period, was given sweetened condensed milk. Diarrhea followed this feeding. He was then given lactic acid milk and did well until one week before admission to the hospital on Oct. 22, 1927. At this time he developed a watery diarrhea and vomiting which persisted. When examined in the hospital he was undernourished and dehydrated. There was slight fever. The ear drums were dull and had a thick appearance, but there was not any definite bulging. Ringer's solution, intraperitoneally, was given on the day of admission, and the following day. Diarrhea, vomiting and dehydration persisted. On October 24, the ear drums were opened, and considerable thin fluid escaped from one ear. Six hours later, pus was seen coming from both ears. More Ringer's solution was given intraperitoneally, and then a blood transfusion was performed. On October 25, marked air hunger developed. Blood was taken for chemical study, and dextrose was given intravenously. Bilateral antrotomy was then performed and extremely large antrums were found filled with milky pus. After operation, and on the following morning, sodium bicarbonate was given intravenously, with temporary relief of air hunger after each administration. During the next three days, diarrhea persisted. More sodium bicarbonate and dextrose solution were given, but no Ringer's solution. Besides whole lactic acid milk with Karo syrup, orange juice in considerable amounts was fed. On October 28, the stools became pasty, and the infant looked much better. From October 29, to November 3, there was a steady gain in weight. The stools, although numerous, were usually small and pasty. On November 3 and 4, diarrhea with fever again occurred. Following this, however, the temperature again became normal and the stools pasty. The patient was discharged from the hospital on November 11, weighing 900 Gm. more than he did on admission.

CASE 19.—Stephen K., an infant, aged 2 months, was breast fed for four weeks and then given whole lactic acid milk feedings. On Oct. 20, 1927, a week before admission to the hospital, he became fretful, vomited, and then developed diarrhea. Bilatral otitis media was found present. Moderate vomiting and diarrhea continued. On October 25, he seemed considerably more toxic. His blood was taken for chemical study and intravenous dextrose was given; on the next day a bilateral antrotomy was performed, and pus and thickening of the mucous membrane were found present. He received a transfusion and made an uneventful recovery.

CASE 20.—Florence M., an infant, aged 8 months, had been breast fed entirely until Oct. 23, 1927, one week before admission to the hospital. Four days before admission, she developed vomiting, diarrhea and high fever. When first examined in the hospital, she was much dehydrated and greatly prostrated. Both ear drums were red, but there was not any bulging. When the ears were opened, they crackled, and only a small amount of serum escaped. Ringer's and dextrose solution were given on October 23 and 24, with marked improvement in the infant's appearance. Pus began to drain from both ears, however. This drainage persisted, together with slight fever, diarrhea and vomiting. Ringer's and dextrose solution were given frequently in an effort to prevent dehydration. On November 3, a bilateral antrotomy was performed, and a considerable amount of pus was found on both sides. After operation, vomiting and diarrhea persisted, and administrations of Ringer's solution and dextrose solution were continued. On November 8, blood was taken for chemical study. Sodium bicarbonate and

dextrose solution were given for two days. On November 11, marked depression of respiration was noted, which was relieved by intravenous administration of calcium chloride. On November 14, carpopedal spasm and convulsive movements of the extremities were noted. These symptoms were relieved by calcium chloride and magnesium sulphate. From that time on, despite normal stools, there was persistent loss in weight. The child's appetite was extremely poor, and when food was forced or gavaged, vomiting occurred. Blood was frequently present in the vomitus. The patient died on November 19, at which time signs of a terminal bronchopneumonia were present.

CASE 21.—George H., an infant, aged 5½ months, was breast fed for three months and then given a good sweet milk formula, on which he did fairly well At the age of five months, he developed what his mother called a "cold." He became feverish, picked at his ears, and had twenty or more green, watery stools daily. He was taken to the St. Louis City Hospital, where he remained for eleven days. During this time, he had considerable vomiting and diarrhea, and frequently intraperitoneal injections of Ringer's solution were given. Antrotomy was advised, and he was transferred to the St. Louis Children's Hospital for operation. On admission, Oct. 26, 1927, he was found to be somewhat undernourished and greatly prostrated. Both ear drums showed openings from which pus was pouring. Both, however, were bulging in the posterior superior quadrants, and there seemed to be tenderness on pressure over the mastoids. Ringer's solution was administered intraperitoneally, and the ear drums were opened more widely. The next day, the respirations were decidedly deeper than normal. Blood was taken for chemical study, and a bilateral antrotomy was performed; pus and excessive granulation tissue were found in each antrum. He was given a transfusion. The next day his appearance was worse, and definite signs of pneumonia were found, from which he died on November 5.

CASE 22.—Dorothy W., an infant, aged 2 months, was breast fed for three weeks; because of failure to gain in weight, she was put on a diet of sweetened condensed milk. On Oct. 26, 1927, six days before admission to the hospital, the patient developed marked watery diarrhea and vomiting. When first examined in the hospital, she was found to be fairly well nourished but considerably dehydrated. A bilateral purulent otitis media was found. Ringer's solution was given intraperitoneally. During the next twenty-four hours, she vomited most of her feedings and had diarrheal stools. When blood was taken for chemical study on October 27, the respirations were deep, and the infant was dehydrated and seemed moribund. A bilateral antrotomy was promptly performed, and pus and thickened membrane were found on each side. Dextrose was then given intravenously, and on October 28, sodium bicarbonate and more dextrose was administered with a decided improvement in the appearance of the child. Vomiting and diarrhea continued, however, and she died on November 1.

CASE 23.—Helen C., an infant, aged 3½ months, had done fairly well on lactic acid milk feedings until Oct. 28, 1927, a week before admission to the hospital, when she developed a respiratory infection with fever, vomiting and diarrhea. When she was admitted, both drums were red, but they did not bulge. Coarse râles were present in the chest. On October 29, both drums were opened, with the release of pus under pressure. Vomiting and diarrhea continued, and considerable fluid was administered parenterally. On October 31, a bilateral antrotomy was performed, and pus and thickened mucous membrane were found on both sides. After operation, signs of pneumonia were noted, and later influenzal meningitis developed. The infant died on November 11.

CASE 24.—Gertrude M., an infant, aged 4 months, was breast fed. She was admitted to the hospital on Oct. 24, 1927, because of a rather mild hot water

bottle burn on the thigh. It was found that she also had a bilateral otitis media and the fever, vomiting and diarrhea which were noted were attributed to that infection. By October 31, her condition had become serious. Fever, vomiting and diarrhea were marked, moderate dehydration was present, and the respirations were of the air hunger type. Considerable Ringer's solution and dextrose were given during the next few days. During this time a large prepectoral subcutaneous abscess developed and was incised. Her condition grew steadily worse, and on November 5, signs of pneumonia were found. A bilateral antrotomy was performed. Pus was found on one side and a thickened membrane on the other. Colon bacilli were cultured from both sides. Her condition became steadily worse, and death occurred on November 6.

CASE 25.—Kenneth B., an infant, aged 5½ months, was fed on sweetened condensed milk and did not do well. On Nov. 1, 1927, a few days before admission to the hospital, he developed vomiting and diarrhea and later pus discharged from his ears. When admitted he was moribund, and died shortly afterward. A bilateral otitis media with probable mastoiditis was present.

CASE 26.—Herbert D., an infant, aged 4 months, from the time he was two weeks of age had been fed on sweetened condensed milk. Moderate vomiting and diarrhea seemed to have been present constantly. On Nov. 5, 1927, when admitted to the hospital, he was undernourished and dehydrated. The temperature was normal. When fed either whole lactic acid milk or thick cereal feeding, vomiting was marked. Watery diarrhea was also present. Considerable Ringer's solution was given parenterally. There was no pus in either ear drum when it was originally opened. On November 9, however, pus was found on both sides. On this day, death occurred from aspiration of vomitus. At autopsy, a bilateral mastoiditis was found, from which hemolytic colon bacilli were cultured.

CASE 27.—Maurice S., an infant, aged 7 months, had been fed chiefly on sweetened condensed milk. On Nov. 10, 1927, about two weeks before admission to the hospital he developed vomiting and diarrhea, which persisted. When admitted, he was moribund, and a bilateral otitis media and probable mastoiditis were present. He died shortly after entrance to the hospital, and blood was taken for chemical study shortly after death. Marked cyanosis preceded death.

## COMMENT

All of the patients described were infants suffering from diarrhea, vomiting, dehydration and "acidosis," and presented a clinical picture similar to that of "alimentary intoxication." In almost all of them, however, clinical evidence of infection was present in the ears and mastoids. In thirteen cases, cultures from the mastoids were obtained at the time of operation. In five of them (cases 4, 5, 18, 24 and 26) colon bacilli were found. These were hemolytic and of the *communior* group. From one case, no. 6, *Bacillus pyocyaneus,* a gram-positive coccus and a gram-positive bacillus were grown. In case 20, *Streptococcus viridans* was found. In the remaining six cases (16, 17, 19, 21, 22 and 23) growth did not occur.

In a large number of the cases (1, 2, 3, 4, 6, 8, 11, 15, 16, 17, 18, 19, 21, 22 and 26) the blood was not examined chemically until after a considerable quantity of salt, in the form of Ringer's solution, had been given intraperitoneally. These cases showed a remarkable chemical picture (table 1; charts 2, 3 and 4). The serum base bicarbonate

*Results of the Chemical Examination of the Blood of Infants with Mastoiditis*

| | | | Blood | | | | | | | | | | | | | | | Therapy | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | (1) | (2) | (3) | (4) | (5) | (6) | (7) | (8) | (9) | (10) | (11) | (12) | (13) | (14) | (15) | (16) | (17) | | (18) | (19) | (20) | (21) |
| | | | | Sodium Chloride | | Carbon Dioxide Content | | Protein | | Inorganic Phosphate | | Lactic Acid | | | | Nonprotein Nitrogen | | | | | | | | |
| Name | Case No. | Date | $p_H$ | Mg. per Cent | BA† Equiv. Cc. N/10 | Per Cent by Volume | BA Equiv. Cc. N/10 | Per Cent | BA Equiv. Cc. N/10 | Mg. per Cent | BA Equiv. Cc. N/10 | Mg. per Cent | BA Equiv. Cc. N/10 | Total Acid Determined, Cc. N/10 | Total Base, Cc. N/10 | Mg. per Cent | BA Equiv. Cc. N/10 | Total BA Equiv. Determined, Cc. N/10 | Freezing Point — C | Date | Ringer's Solution, Cc. | Dextrose, 10-20% Cc. | Blood, Cc. | Sodium Bicarbonate 5% Cc. |
| Curtis T..... | 1 | 10/ 6/24 | .... | 712 | 121 | 32.9 | 13.9 | 5.55 | 9.5 | .... | .... | .... | .... | 144 | ... | 130 | 23.2 | 167 | ... | 10/4 to 10/6 | 700 | 200 | 230 | .. |
| Frank S..... | 2 | 10/27/24 | .... | 837 | 143 | 20.9 | 8.9 | 5.06 | 8.6 | .... | .... | 41.5 | 4.6 | 165 | ... | 91.0 | 16.2 | 191 | ... | 10/9 to 10/27 | 6,150 | 1,370 | 100 | .. |
| Minnie S..... | 3 | 10/30/24 | .... | 725 | 124 | 31.2 | 13.2 | 6.55 | 11.1 | .... | .... | 25.2 | 2.9 | 151 | ... | 110 | 19.6 | 171 | ... | 10/31 to 11/3 | 500 | ..... | 100 | .. |
| | | 10/31/24 | .... | 655 | 112 | 33.8 | 13.7 | .... | .... | .... | .... | 17.7 | 2.0 | 128 | ... | 68.0 | 12.1 | | | | | | | |
| Kenneth H..... | 4 | 1/14/25 | .... | 724 | 124 | 14.5 | 6.2 | .... | .... | .... | .... | 63.8 | 9.1 | 139 | ... | .... | .... | ... | ... | 1/12 to 1/17 | 1,300 | 270 | ... | 50 |
| | | 1/15/25 | .... | 748 | 128 | 29.8 | 12.6 | 4.81 | 8.2 | .... | .... | 50.5 | 5.6 | 144 | | | | | | | | | | |
| John G..... | 5 | 3/31/25 | 7.53 | 526 | 90 | 45.0 | 19.0 | .... | .... | 6.0 | 3.5 | .... | .... | 113 | ... | .... | .... | ... | ... | 3/31 to 4/22 | 3,270 | 470 | 200 | .. |
| | | 4/ 9/25 | .... | 584 | 100 | 37.4 | 15.8 | .... | .... | 4.3 | 2.8 | .... | .... | 119 | | | | | | | | | | |
| | | 4/22/25 | 7.00 | 714 | 122 | 9.8 | 4.1 | .... | .... | .... | .... | .... | .... | 126 | ... | 113 | 21.1 | | | | | | | |
| Leo S..... | 6 | 5/21/25 | .... | 902 | 154 | 12.2 | 5.6 | .... | .... | .... | .... | .... | .... | 160 | ... | 93.0 | 17.1 | ... | ... | 4/23 to 5/21 | 3,000 | 290 | 255 | .. |
| Selma G..... | 7 | 6/19/25 | .... | 602 | 103 | 17.6 | 7.5 | .... | .... | .... | .... | .... | .... | 111 | ... | 106 | 18.9 | ... | ... | 6/18/25 | 500 | 150 | ... | .. |
| Mary G..... | 8 | 7/10/25 | 7.25 | 704 | 120 | 18.4 | 7.8 | .... | .... | 9.6 | 5.5 | .... | .... | 133 | ... | 106 | 18.9 | ... | ... | 7/8 to 7/15 | 550 | 225 | 75 | .. |
| | | 7/12/25 | .... | 590 | 101 | 15.6 | 6.6 | .... | .... | 11.7 | 6.8 | .... | .... | 114 | ... | 122 | 21.8 | | | | | | | |
| | | 7/13/25 | 7.26 | 568 | 97 | 14.4 | 6.1 | .... | .... | .... | 6.8* | .... | .... | 103 | ... | 148 | 26.5 | | | | | | | |
| Mildred R..... | 9 | 7/10/25 | 7.38 | 656 | 112 | 34.0 | 14.4 | .... | .... | 5.7 | 3.3 | .... | .... | 130 | ... | 40.0 | 7.1 | ... | ... | 7/10 to 7/15 | 625 | 75 | 265 | .. |
| | | 7/13/25 | 7.30 | 596 | 102 | 24.0 | 10.3 | 5.36 | 9.1 | 4.0 | 2.3 | .... | .... | 124 | ... | 41.0 | 7.3 | | | | | | | |
| | | 7/15/25 | .... | 683 | 117 | 36.7 | 15.6 | 5.68 | 9.7 | 2.8 | 1.7 | .... | .... | 144 | ... | 50.0 | 8.9 | | | | | | | |
| Tommie C..... | 10 | 7/20/25 | 6.90 | 532 | 91 | 21.1 | 9.0 | 8.71 | 12.5 | 10.9 | 6.3 | .... | .... | 119 | ... | 44.0 | 7.8 | 128 | ... | 7/20 to 7/25 | 2,650 | 525 | 200 | . |
| | | 7/23/25 | 6.95 | 785 | 134 | 25.6 | 10.9 | 8.49 | 12.4 | 18.5 | 10.7 | .... | .... | 168 | ... | 76.0 | 13.6 | 182 | | | | | | |
| | | 7/25/25 | 7.05 | 831 | 142 | 14.0 | 5.9 | 7.20 | 10.2 | 15.8 | 9.2 | .... | .... | 175 | ... | 125 | 22.3 | 197 | | | | | | |
| Dorothy B..... | 11 | 9/21/25 | 7.29 | 703 | 120 | 27.6 | 11.7 | .... | .... | .... | .... | .... | .... | 132 | ... | 62.0 | 11.1 | 143 | ... | 9/18 to 9/22 | 850 | 200 | 100 | .. |
| | | 9/22/25 | 7.42 | 714 | 122 | 31.0 | 13.1 | 7.85 | 14.0 | 4.0 | 2.3 | .... | .... | 151 | ... | 53.0 | 9.5 | 161 | | | | | | |

| | | | | | | | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Robert U..... | 12 | 12/27/25 | .... | 714 | 122 | 42.7 | 18.1 | .... | .... | .... | .... | .... | .... | 140 | ... | .... | .... | ... | ... | 12/27 to 1/15 | 125 | 300 | 200 | .. |
| | | 12/28/25 | 7.40* | 729 | 125 | 46.8 | 19.8 | 8.60 | 15.4 | .... | .... | .... | .... | 160 | ... | 67.0 | 12.0 | 182 | | | | | | |
| | | 1/ 2/26 | 7.60* | 560 | 95.7 | 139 | 50.0 | 7.42 | 14.1 | .... | .... | .... | .... | 169 | | | | | | | | | | |
| | | 1/ 4/26 | 7.50* | 644 | 110 | 73.0 | 31.0 | 6.98 | 12.8 | .... | .... | .... | .... | 154 | ... | 80.0 | 14.3 | 168 | | | | | | |
| | | 1/11/26 | 7.45 | 562 | 96.0 | 60.2 | 25.5 | 6.34 | 11.4 | 7.8 | 4.5 | .... | .... | 137 | ... | 27.0 | 4.8 | 142 | | | | | | |
| | | 2/ 3/26 | .... | 491 | 83.7 | 56.2 | 23.8 | .... | .... | .... | .... | .... | .... | 108 | ... | 31.4 | 5.6 | | | | | | | |
| Louis B..... | 13 | 6/15/26 | 7.20 | 685 | 117 | 20.7 | 8.8 | 7.20 | 11.8 | .... | .... | 45.2 | 5.0 | 142 | ... | 97.2 | 17.3 | 159 | ... | 6/14 to 6/17 | 1,200 | 80 | 150 | .. |
| Marguerite G... | 14 | 9/ 2/26 | 7.08 | 519 | 88.8 | 15.0 | 6.4 | .... | .... | .... | .... | .... | .... | 95 | ... | 120 | 21.4 | ... | ... | 9/1/26 | 250 | ..... | ... | .. |
| Isabelle G..... | 15 | 10/26/26 | 7.00 | 788 | 135 | 8.2 | 3.5 | 7.85 | 11.7 | .... | .... | .... | .... | 150 | ... | 69.6 | 12.4 | ... | ... | 10/25 to 11/1 | 600 | 60 | 75 | 125 |
| | | 11/ 1/26 | 7.06 | 705 | 120 | 15.3 | 6.5 | 7.85 | 12.1 | .... | .... | .... | .... | 139 | | | | | | | | | | |
| Bonnie S..... | 16 | 9/24/27 | .... | 819 | 140 | 28.5 | 12.1 | .... | .... | .... | .... | .... | .... | 152 | ... | .... | .... | ... | ... | 9/22 to 9/24 | 850 | 125 | 60 | .. |
| Denise C..... | 17 | 10/21/27 | 7.00 | 750 | 128 | 14.5 | 6.1 | 7.42 | 11.1 | 8.9 | 5.2 | .... | .... | 150 | ... | .... | .... | ... | ... | 10/18 to 10/21 | 1,450 | 270 | 75 | .. |
| Salvatore R..... | 18 | 10/25/27 | 7.13 | 802 | 137 | 17.1 | 7.2 | 8.92 | 14.1 | 7.9 | 4.6 | 68.0 | 7.5 | 170 | 162 | 60.0 | 10.7 | 181 | ... | 10/22 to 10/26 | 1,600 | 100 | 90 | 100 |
| | | 10/26/27 | 7.30 | 714 | 122 | 23.5 | 10.0 | 7.20 | 12.2 | 6.9 | 4.0 | 64.2 | 7.1 | 155 | 157 | 61.0 | 10.9 | 168 | ... | 10/26/27 | ..... | 100 | ... | 50 |
| | | 10/27/27 | 7.27 | 607 | 114 | 24.5 | 10.4 | 7.42 | 12.4 | 7.7 | 4.5 | 37.8 | 4.2 | 146 | 155 | 85.0 | 15.2 | 170 | 550 | 10/27 | ..... | 100 | ... | 50 |
| | | 10/28/27 | 7.30* | 591 | 101 | 31.0 | 13.2 | 6.77 | 11.5 | .... | 4.5* | .... | 3.6* | 126 | 143* | 76.0 | 13.5 | 157* | ... | 10/28 | ..... | 50 | ... | 50 |
| | | 11/ 4/27 | 7.28 | 645 | 110 | 29.0 | 12.3 | 7.20 | 12.2 | 8.0 | 4.6 | 26.5 | 3.0 | 142 | 136 | 50.0 | 8.9 | 151 | | | | | | |
| | | 11/11/27 | 7.39 | 591 | 101 | 51.5 | 21.8 | 6.34 | 11.2 | .... | 4.0* | .... | 2.5* | 141 | 137 | 42.0 | 7.5 | 140 | | | | | | |
| Stephen K..... | 19 | 10/25/27 | 7.30 | 679 | 116 | 34.4 | 14.6 | 5.68 | 9.7 | 5.2 | 3.0 | 25.2 | 2.8 | 146 | 150 | 26.0 | 4.6 | 155 | ... | To 10/25 | 600 | 70 | ... | .. |
| Florence M..... | 20 | 10/27/27 | 7.45 | 585 | 100 | 54.5 | 23.2 | 5.25 | 9.5 | 3.5 | 2.0 | 17.6 | 2.0 | 137 | 149 | 16.0 | 2.9 | 152 | 585 | 10/23 to 10/27 | 1,250 | 200 | ... | .. |
| | | 11/ 8/27 | 7.33 | 734 | 125 | 33.7 | 14.3 | 5.47 | 9.4 | 3.8 | 2.2 | 15.1 | 1.7 | 153 | 150 | 46.6 | 8.0 | 167 | 582 | 10/27 to 11/ 8 | 4,700 | 1,380 | ... | .. |
| | | 11/ 9/27 | 7.33 | 731 | 125 | 38.0 | 16.1 | 6.77 | 11.6 | .... | 2.1* | .... | 2.0* | 153 | ... | .... | 8.0* | 165* | ... | 11/8 | 450 | 150 | ... | .. |
| | | 11/11/27 | 7.47 | 632 | 108 | 69.3 | 28.4 | 4.81 | 8.7 | 3.5 | 2.0 | .... | 2.0* | 147 | ... | .... | 6.0* | 155* | ... | 11/9 to 11/11 | ..... | 300 | ... | 100 |
| | | 11/14/27 | 7.48 | 580 | 99.2 | 65.0 | 27.5 | 4.59 | 8.4 | .... | 2.0* | .... | 2.0* | 135 | ... | .... | 5.0* | ... | ... | 11/11 to 11/14 | ..... | 380 | ... | .. |
| George H..... | 21 | 10/27/27 | 7.35 | 712 | 122 | 35.1 | 14.9 | 5.30 | 9.2 | 3.0 | 1.7 | 31.5 | 3.5 | 151 | 156 | 16.0 | 2.9 | 150 | ... | To 10/27 | 400+ | ..... | ... | .. |
| Dorothy W..... | 22 | 10/27/27 | 7.25 | 674 | 115 | 21.2 | 9.0 | 7.42 | 12.3 | 5.0 | 2.9 | 18.9 | 2.1 | 146 | 140 | 48.0 | 8.6 | 149 | ... | 10/26 to 10/27 | 700 | ..... | ... | .. |
| | | 10/28/27 | 7.28 | 675 | 115 | 20.8 | 8.8 | 6.98 | 11.8 | .... | .... | .... | .... | 136 | ... | .... | .... | ... | ... | To 10/28 | ..... | 200 | ... | .. |
| | | 10/31/27 | 7.39 | 632 | 108 | 36.4 | 15.4 | 7.85 | 13.9 | .... | .... | .... | .... | 137 | ... | .... | .... | ... | ... | To 10/31 | ..... | 600 | 50 | .. |
| Helen C..... | 23 | 10/29/27 | 7.38 | 597 | 102 | 40.5 | 17.2 | 5.25 | 9.2 | .... | .... | .... | .... | 128 | 151 | 22.0 | 3.9 | 155 | 550 | To 10/29 | 800 | ..... | ... | .. |
| | | 11/10/27 | 7.20 | 772 | 132 | 17.0 | 7.2 | 6.55 | 10.7 | .... | .... | 25.2 | 2.8 | 153 | 152 | 31.0 | 5.5 | 158 | ... | To 11/10 | 4,425 | 2,080 | ... | .. |
| Gertrude M..... | 24 | 10/31/27 | 7.37 | 585 | 100 | 45.2 | 19.1 | 6.77 | 11.8 | 5.5 | 3.2 | 29.0 | 3.2 | 137 | 146 | 20.0 | 3.6 | 150 | ... | To 10/31 | 450 | 180 | ... | .. |
| Kenneth B..... | 25 | 11/ 1/27 | .... | 550 | 94.2 | 46.7 | 19.7 | | | | | | | | | | | | | | | | | |
| Herbert D..... | 26 | 11/ 8/27 | 7.28 | 854 | 146 | 24.8 | 10.5 | 6.77 | 11.4 | 4.0 | 2.4 | 15.1 | 1.7 | 172 | 175 | 34.0 | 6.1 | 181 | 620 | 11/5 to 11/8 | 2,250 | 200 | ... | .. |
| Maurice S..... | 27 | 11/11/27 | 7.08 | 544 | 93 | 22.2 | 9.4 | 9.56 | 14.8 | 11.5 | 6.7 | 108 | 12.0 | 146 | 139 | 64.0 | 11.4 | 150 | 572 | | | | | |

* Assumed values.

† Acid-base equivalent.

content in harmony with the observations of Howland and Marriott,[11] and Schloss and Stetson,[12] and $p_H$ were much reduced, while the base chloride content was frequently greatly increased. In one case (case 6) the increase amounted to 54 per cent, making the base chloride content alone equal to the total electrolyte content encountered in normal infants. The protein content was usually slightly above the value for normal infants, but occasionally was low. Acetone or diacetic acid[13] was not present in significant amounts in any case. Frequently lactic acid was increased in cases of anhydremia, as noted by Clausen,[14] but it was also frequently normal. The highest lactic acid value was found in case 27; this alone accounted for 86 per cent of the reduction of the base bicarbonate present. On the other hand, in case 22, in which a 61 per cent reduction of base bicarbonate was found, the lactic acid content was normal. Phosphoric acid was frequently increased to two or three times its normal value, but generally accounted for only a small fraction of the reduction in base bicarbonate. In seven of the fifteen instances in which the total base was determined, the base showed good agreement with the total acid determined, plus the normal amount of undetermined acid (R). In the remaining, the total amount of fixed base was less than the total amount of acid, a rather surprising observation, and one which we cannot certainly attribute to experimental inaccuracy. In practically all of the cases, in keeping with the observations of Schloss,[15] the protein nitrogen was considerably increased. Dextrose also was usually slightly increased.

A natural result of these changes (increase of all salts except base bicarbonate, and increase of the principal nonelectrolyte crystalloids, urea and dextrose) would be a considerable increase in osmotic pressure. It is to be regretted that so few freezing points were made of the specimens of blood showing the largest increase of osmotically active substances. The most striking instance is found in case 26.

In those cases in which the blood was examined before significant amounts of fluid were administered parenterally (cases 5, 10, 20, 23, 24

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11. Howland, J., and Marriott, W. McK.: Acidosis Occurring with Diarrhea, Am. J. Dis. Child. **11**:309 (May) 1916.

12. Schloss, O. M., and Stetson, R. E.: The Occurrence of Acidosis with Severe Diarrhea, Am. J. Dis. Child. **13**:218 (March) 1917.

13. The total amount of acetone bodies in the blood was never quantitatively determined. In the lactic acid determinations, however, the volatile substances in the filtrate which ordinarily are driven off in the short distillation previous to oxidation by potassium permanganate were made to pass through Scott-Wilson reagent, as a qualitative test for acetone.

14. Clausen, S. W.: Anhydremic Acidosis Due to Lactic Acid, Am. J. Dis. Child. **29**:761 (June) 1925.

15. Scloss, O. M.: Intestinal Intoxication in Infants, Am. J. Dis. Child. **15**:165 (March) 1918.

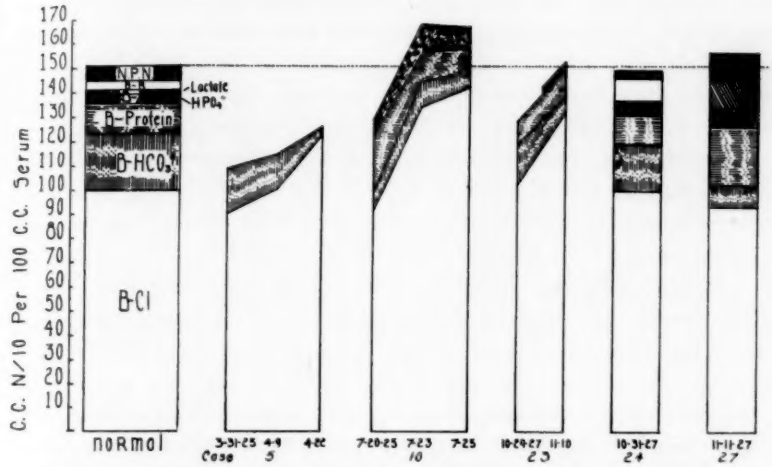


Chart 1.—Electrolyte and nonprotein nitrogen of blood serum in cases of mastoiditis with diarrhea, vomiting, dehydration and oliguria, before and after administration of salt solution.

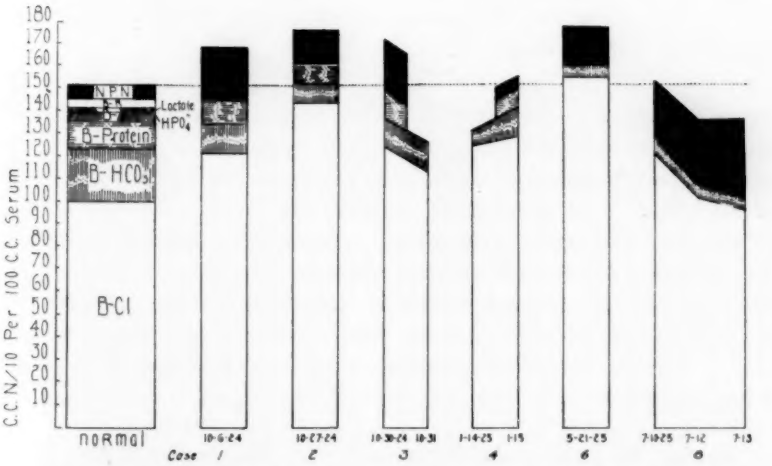


Chart 2.—Electrolyte and nonprotein nitrogen of blood serum in cases of mastoiditis with diarrhea, vomiting, dehydration and oliguria, after administration of large amounts of salt solution.

and 27 of table 1, and charts 1 and 4) a somewhat different chemical picture was present; the base bicarbonate, although reduced, was not usually as low, while the base chloride was also reduced. A significant difference did not exist in the concentrations of the other substances studied. In these cases, consequently, the total electrolyte content was frequently considerably reduced. Usually, however, the increase in nonprotein nitrogen was sufficient to maintain a normal total osmotically active content and freezing point.

In attempting to explain such observations as these, it seems best first to consider which changes could be attributed to the chief symptoms present, vomiting, diarrhea, dehydration and oliguria. When vomiting occurs in an otherwise healthy infant because of pyloric stenosis,[3] the result is a loss of hydrochloric acid, base chloride and water from the body. The loss of hydrochloric acid far exceeds the loss of base chloride and leads to the retention of base bicarbonate. In the cases in which infection is present, the effects of vomiting would certainly be expected to be in the same direction. Because of the fact, however, as shown by Marriott,[16] that considerably less hydrochloric acid is secreted into the stomach after feeding when infants have infection, one would not expect, as a rule, as great a reduction in the chloride content of the blood when vomiting resulted from infection as when it resulted from obstruction.[17] In harmony with this assumption is the fact that in those cases studied before the administration of salt solution, the chloride content of the serum was definitely, though only moderately, reduced. In those cases, however, the base bicarbonate content was not increased, but actually reduced, sometimes extremely so. Obviously, then, vomiting could not have been the only factor that produced the changes observed.

In considering the cause for the reduction of base bicarbonate in the presence of a reduction of base chloride, there seem to be but two possible explanations; either acids other than hydrochloric acid have displaced the bicarbonate ion, or base bicarbonate has actually been lost from the body. As already mentioned, acetone bodies were absent, and lactic acid and phosphoric acid seldom accounted for the major portion of the reduction of base bicarbonate observed. Increase of protein was seldom large enough (except in case 27, which also showed a high lactic acid value) to be considered from the standpoint of its base-finding capacity. One is forced to conclude, therefore, that retention of the acid was not the cause of the reduction of base bicarbonate unless some

16. Marriott, W. McKim, and Davidson, L. T.: The Acidity of the Gastric Contents of Infants, Am. J. Dis. Child. **26**:542 (Dec.) 1923.

17. That pyelitis can occasionally cause as marked reduction in plasma chloride as pyloric stenosis has, however, been observed (Hartmann and Smythe, footnote 2).

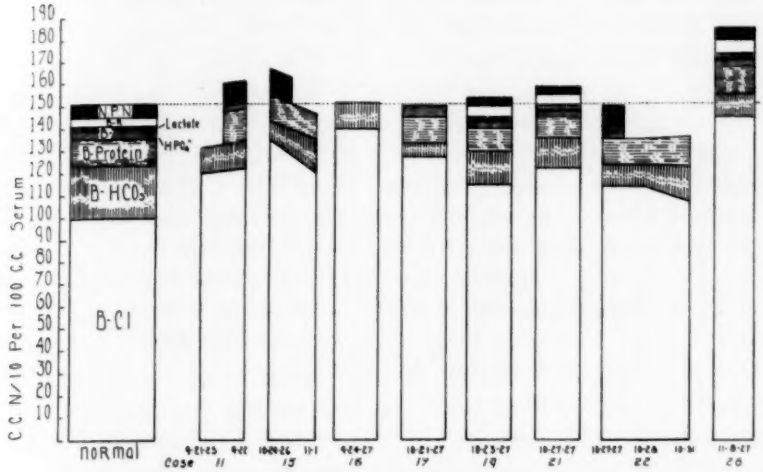


Chart 3.—Electrolyte and nonprotein nitrogen of blood serum in cases of mastoiditis with diarrhea, vomiting, dehydration and oliguria, after administration of large amounts of salt solution.

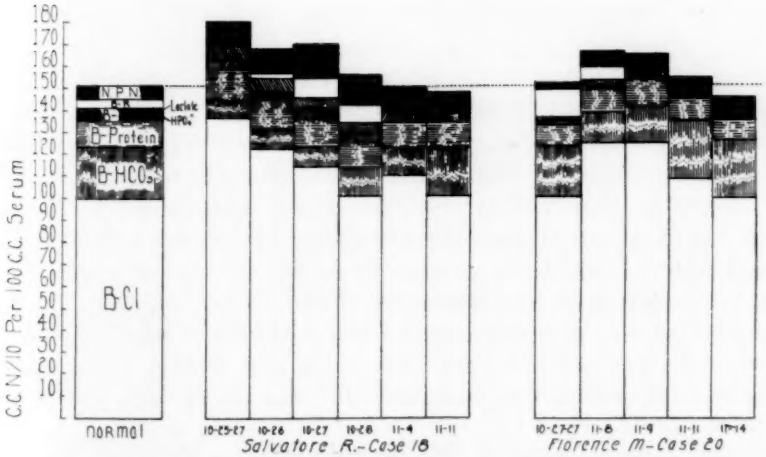


Chart 4.—Electrolyte and nonprotein nitrogen of blood serum in cases of mastoiditis with diarrhea, vomiting, dehydration and oliguria, before and after administration of salt solution.

unidentified acid were responsible. Against this possibility, however, is the failure to find any marked excess of total base over total determined acid. It seems most reasonable to conclude, therefore, that the reduction of base bicarbonate resulted chiefly from loss from the body.

Because of the pronounced oliguria, it is reasonable to suppose that this loss of base bicarbonate could not have occurred by way of the urine, and the highly acid reaction $p_H$ 5 of the urine in the few instances observed makes the possibility still less likely. By exclusion, therefore, even in the absence of the analysis of the stool, it seems much more likely that the base bicarbonate was lost by way of the intestinal tract, originally probably having been withdrawn from the blood in the secretion of pancreatic juice, which normally is rich in base bicarbonate and of considerable volume. This conclusion seems further justified by the work of Holt, Courtney and Fales[18] and of Meyer,[19] who have found a considerable excess of fixed base over fixed acid in diarrheal stools.

Besides loss of hydrochloric acid, base chloride and base bicarbonate by way of vomitus and stool, large amounts of water were lost, which resulted in dehydration. The occasional decided increase of serum protein presumably resulted from loss of water from the blood. In turn, this dehydration caused the oliguria and the increase of nonprotein nitrogen and phosphoric acid due to incomplete elimination by the kidney. It is to be noted in case 18, however, that the nonprotein nitrogen of the blood remained definitely elevated for a considerable period after a normal water balance and urinary output had been established. Dehydration probably also accounted for the increase in the lactic acid observed, through its effect in diminishing the volume and flow of the blood, with consequent failure of the circulation to provide the tissues with sufficient oxygen to prevent the accumulation of lactic acid.

From these considerations, it seems reasonable to explain all the chemical changes encountered before the administration of salt solution as direct results of vomiting, diarrhea, loss of water and renal insufficiency due to oliguria. If the same factors remain at work during the time that large quantities of salt solution are being given, we might expect continued loss of water with continued dehydration and its direct consequences, continued loss of base bicarbonate by way of the intestinal tract, but replacement of chloride lost in the vomitus and stool by the large parenteral administration of chloride containing Ringer's solution; and if relatively more water than salt of the salt solution administered is lost, one might expect an increase of the base chloride content of the

18. Holt, L. M.; Courtney, A. M., and Fales, H. L.: The Chemical Composition of Diarrheal as Compared with Normal Stools in Infants, Am. J. Dis. Child. **9**:213, 1915.

19. Meyer, L. F.: Ueber den Stoffewechsel der Alimentären Dekomposition, Jahrb. f. Kinderh. **71**:389, 1910.

serum to considerably above the normal. This is what seems actually to have occurred. As long as infection continued, the gastro-intestinal symptoms persisted, with its direct results on the chemical constitution of the body. The effect of this chloride increase, furthermore, would be to produce just what has been observed, a still further reduction of the base bicarbonate content of the blood because of the reciprocal way with which the chloride and bicarbonate ions seem to vary.

If these explanations are correct, the logic of the usual type of therapy for such cases as these is questionable. As can be noted from the report of cases and table of data, the earlier policy of handling these cases was as follows:

Besides local treatment directed toward establishing good drainage from an infected focus (such as paracentesis of the ear drum and antrotomy), an effort was made to administer large amounts of fluid, as Ringer's solution, intraperitoneally. In this way, it was hoped to prevent anhydremia and lactic acid acidosis, and to promote diuresis, principally with the idea of washing out as much toxin absorbed from an infected focus as possible. The actual results have already been mentioned; as long as the infection persisted, vomiting, diarrhea and loss of water continued, urinary secretion remained scanty, and although lactic acid accumulation in dangerous amounts was frequently prevented, hydrochloric acid in sometimes tremendous and presumably fatal amounts was substituted.

After this conclusion was forced on us, it seemed evident that salt solution administration in large amounts when diarrhea and oliguria persisted was strongly contraindicated; but a large intake of water remained necessary. In addition, since there almost certainly occurred an actual loss of base bicarbonate from the body, and not simply an accumulation of easily oxidizable acid which could suddenly release the base to which it had been bound, it seemed wise to administer sodium bicarbonate intravenously in amounts calculated to increase the base bicarbonate content of the blood and tissue fluid slowly but effectively. As can be noted in cases 18 and 20, when the administration of saline solution was discontinued, and when water was administered by mouth and dextrose and sodium bicarbonate was injected intravenously, sudden prompt improvement occurred, which permitted eventual recovery.

It is especially to be noted in case 18, however, that while diarrhea continued, the prompt improvement after intravenous administration of sodium bicarbonate was temporary, and that in a short time the base bicarbonate content again returned to a low value. During a period of seventy-two hours, 12.5 Gm. of sodium bicarbonate was given intravenously and a considerable, if unmeasured, amount of orange juice by mouth, which would make a total at least twice as great as the entire normal content of the body. Despite this large amount of administered

sodium bicarbonate, alkalosis did not result. In case 20, however, diarrhea ceased almost simultaneously with the administration of sodium bicarbonate in an amount calculated to be somewhat larger than that lost from the body. In this case, alkalosis with tetany developed. The necessity for care in the administration of sodium bicarbonate was thus clearly emphasized.

From all that has previously been discussed, it seems reasonably certain that the changes in the chief plasma constituents encountered in cases of mastoiditis in infancy result from the chief symptoms of that infection; namely, vomiting, diarrhea, dehydration and oliguria. The exact means by which infection so far from the gastro-intestinal tract produces such symptoms is still much in doubt. In connection with this question, however, one striking fact is observed, namely, that the clinical picture of these infants resembled closely that in cases previously described as "alimentary intoxication." One naturally asks the question "Were the latter cases really cases of unrecognized mastoiditis?" Another explanation, however, is more probable. One theory of alimentary intoxication is that food is split into toxic fractions by the colon bacillus which has invaded high into the small intestine. As the result of absorption of these products, vomiting, diarrhea and dehydration occur. In this connection, pointing possibly to the nature of such substances, it has been shown by Hanke and Koessler [20] that certain strains of colon bacilli can convert the amino-acids, histidine and tyrosine, into such extremely toxic products as histamine and tyramine. Absorption of such toxic amines into the general circulation would lead to vomiting, diarrhea and shock—a picture common to cases of "alimentary intoxication" and of mastoiditis. If this can occur from the intestinal tract, as would seem possible in the case of the young infant, despite the apparently highly developed detoxifying capacity of the intestinal mucous membrane, one might reasonably expect it to occur also from the mastoid. It is hoped that investigation being carried out along this line will throw more light on this important question.

## SUMMARY AND CONCLUSIONS

As a result of the study of the chemical changes that occurred in the blood in twenty-seven infants with mastoiditis which resembled clinically "alimentary intoxication," it was found that:

1. The chemical changes are due to the chief symptoms of the infection: vomiting, diarrhea, dehydration and oliguria.

(*a*) As a result of vomiting, hydrochloric acid and base chloride are lost from the body. The loss of hydrochloric acid is not compensated for by base bicarbonate, as in cases of vomiting due to pyloric stenosis.

20. Hanke, M. T., and Koessler, K. K.: Studies on Proteinogenous Amines, J. Biol. Chem. **59**:855, 1924.

(*b*) Base bicarbonate is much reduced in the body, not so much because of the increase of acid, but because of actual loss from the body by way of the intestinal secretion and diarrheal stool.

(*c*) Dehydration accounts for the occasional increase of lactic acid and protein.

(*d*) Oliguria accounts for the increase of phosphoric acid and non-protein nitrogen in the blood.

2. The administration of large amounts of saline solution when diarrhea and oliguria persist is contraindicated because of the marked tendency toward retention of base chloride, which tends to keep base bicarbonate reduced, producing extreme persistent and often fatal acidosis.

3. Water in large quantity must be given orally as such, or intravenously as dextrose solution.

4. Because of the frequent marked loss of base bicarbonate from the body, intravenous administration of sodium bicarbonate is often indicated, and may at times be a life-saving measure.

5. These important measures (administration of water, dextrose and sodium bicarbonate) must be carried out in conjunction with the local surgical treatment (antrotomy) of the infected focus, and must be continued as long as diarrhea, vomiting, dehydration and acidosis persist.

# MINERAL METABOLISM ON A HIGH MINERAL DIET *

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In the feeding of infants with milk modifications, what amounts and what proportions of the mineral elements should be in the formula? As in many other respects in infant nutrition, empiricism has striven to imitate nature by making the salt content of milk modifications approximate that of human milk. Although no formula superior to breast milk has been devised for the average baby, it does not follow that breast milk contains the ideal composition of salts for sick babies or for normal babies at all times.

It is well known that in the present civilization, infants nourished at the breast are subject to such defects in mineral metabolism as are expressed in rickets and tetany. Premature infants are especially subject to rickets, and practically all infants develop this condition, whether breast feeding or milk modifications are employed. The concentration of salts in cow's milk is about three times that of breast milk. A tacit assumption has been made, especially by the German investigators, that this component of cow's milk represents an excess above the infant's requirements and is a "luxus" consumption.

The present study was made on an infant who was fed in one period skimmed cow's milk plus cane sugar with salts added so as to make the concentration of each mineral element approximately double that of cow's milk. The same food without salt additions was given in a second period. Excretion in the urine and feces was followed, and a balance of each of the minerals was computed. The data have been calculated in terms of grams of the particular elements, and also in terms of cubic centimeters of tenth-normal acid or alkali, so that we were able to compute the acid-base balance during the periods. The methods were the same as those recorded in a previous study,[1] and the computation of base balances has also been previously described.[1]

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* From the Departments of Pediatrics and Physiological Chemistry, Yale University of New Haven, Conn.

1. Shohl, A. T., and Sato, A.: Acid-Base Metabolism: I. Determination of Base Balance, J. Biol. Chem. **58**:235, 1923.

### REPORT OF A CASE

*History.*—A boy baby, the larger of normal full term twins of the sixth pregnancy, born July 27, 1924, received milk modifications after two weeks of breast feeding. The birth weight was not known. The baby had not had any disease until a few days before his first admission to the hospital on Nov. 25, 1924. The diagnosis was otitis media and indigestion. After eighteen days in the hospital, he was "discharged against advice," but was readmitted on January 22 with bronchopneumonia and rickets of prematurity. He weighed 4.7 Kg. The child thrived on sour milk modifications of high caloric value, from 120 to 219 calories per kilogram, ultraviolet radiation and 8 cc. of cod liver oily daily. The laboratory observations did not show anything of interest. Four days before the experimental period, he was given a diet equal to 136 calories per kilogram, consisting of 1 liter of skimmed milk plus ten per cent of cane sugar, plus extra salts. On March 9, at the age of 8 months, when he weighed 5.9 Kg., he was placed on the metabolism bed and the same diet was continued. He remained for four days, gaining 75 Gm. daily. He weighed 6.2 Kg. when the first period ended. The marked stool was recovered the next day. He was then given the same food without salt additions. Ten days later, the second study was performed, from March 23 to 28. During this time, his weight fell from 6.5 to 6.4 Kg. The serum calcium was 10 mg. per hundred cubic centimeters and the inorganic phosphorus 7.1 mg. per hundred cubic centimeters at the end of the first period. Ten days later, the calcium was the same and the phosphorus 5.9 mg. He was discharged in good condition shortly after the experiment was terminated.

This child could not be considered normal, but he had recovered from pneumonia more than six weeks before the first experimental period. He had received active treatment for rickets for some months, and the values of his serum calcium and inorganic phosphorus were normal according to the laboratory observations. The case is not offered as an example of normal metabolism, because the infant lost weight during the second period of observation. The important point is the comparison of the metabolism of the infant during the periods of diet with and without added salt.

### PROGRESS OF EXPERIMENT

The experiment proceeded without known errors; all specimens were saved and vomiting did not occur. The infant was in excellent condition when removed from the bed after each period. The stools were slightly more voluminous during the added salt period. The dry weight averaged 14.6 Gm. for the milk period and 16.9 Gm. when extra salts were added. The volumes of urine averaged 346 cc. and 462 cc. for the respective periods.

The data are given in tables 1 and 2. The results are expressed in table 1 in grams of each element to show its absolute and percentage retention. In table 2, the same data are expressed in terms of tenth-normal solutions. This affords a direct comparison of elements, and by the reckoning of acids and bases one can measure the "base balance" and the "salt balance."

Analysis of the results show that when twice the amount of salt was given, more of each element was retained. In the period when

additional salts were given, not only were more alkalis retained, but also more acids, so that the excess of base over acid and the composition of the retained material does not differ essentially from normal. In both periods, a fairly constant proportion of intake of each element was excreted by way of the urine and stool.

TABLE 1.—*Mineral Metabolism Expressed in Grams*

| | Food: Period* | | Feces: Period | | Urine: Period | | Total: Period | | Balance: Period | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | | | | | I | | II | |
| | I Gm. | II Gm. | I Gm. | II Gm. | I Gm. | II Gm. | I Gm. | II Gm. | Gm. | % | Gm. | % |
| Ca | 3.11 | 1.55 | 2.23 | 1.10 | 0.09 | 0.01 | 2.24 | 1.11 | 0.87 | 28.0 | 0.44 | 28.3 |
| Mg | 0.22 | 0.11 | 0.21 | 0.11 | † | † | 0.21 | 0.11 | 0.01 | 5.3 | 0.00 | 1.0 |
| Na | 1.80 | 0.69 | 0.05 | 0.05 | 1.32 | 0.55 | 1.37 | 0.60 | 0.43 | 24.0 | 0.09 | 14.0 |
| K | 3.91 | 1.96 | 0.14 | 0.10 | 2.88 | 1.65 | 3.02 | 1.75 | 0.89 | 22.1 | 0.21 | 10.7 |
| P | 3.07 | 1.32 | 1.32 | 0.47 | 1.26 | 0.59 | 2.58 | 1.06 | 0.49 | 16.0 | 0.26 | 20.0 |
| S | 0.86 | 0.48 | † | † | 0.44 | 0.37 | 0.44 | 0.37 | 0.42 | 49.0 | 0.11 | 23.0 |
| Cl | 3.80 | 1.56 | 0.03 | 0.10 | 2.97 | 1.35 | 3.00 | 1.45 | 0.80 | 21.0 | 0.11 | 7.0 |

* Period I in these tables denotes a period in which the diet consisted of 1 liter of skimmed milk and 100 Gm. cane sugar, with salts added so as to make the amount of each mineral element approximately double that in skimmed cow's milk; period II, the administration of the same food without salt additions.

† Analyses not made.

TABLE 2.—*Mineral Metabolism Expressed in cc. of 0.1 Normal Solution*

| | Food: Period | | Feces: Period | | Urine: Period | | Total: Period | | Balance: Period | |
|---|---|---|---|---|---|---|---|---|---|---|
| | I | II | I | II | I | II | I | II | I | II |
| | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N | Cc. 0.1 N |
| Ca | 1,548 | 774 | 1,115 | 501 | 5 | 6 | 1,120 | 507 | 428 | 267 |
| Mg | 189 | 95 | 177 | 93 | 0 | 0 | 177 | 93 | 12 | 2 |
| Na | 784 | 302 | 23 | 32 | 573 | 239 | 596 | 271 | 188 | 31 |
| K | 1,000 | 500 | 36 | 27 | 738 | 423 | 774 | 450 | 226 | 50 |
| − Bases (A) | −3,521 | −1,671 | −1,351 | −653 | −1,316 | −668 | −2,667 | −1,321 | −854 | −350 |
| $PO_4$ | 1,780 | 770 | 768 | 273 | 732 | 324 | 1,500 | 507 | 280 | 173 |
| $SO_4$ | 535 | 297 | † | † | 250 | 204 | 250 | 204 | 285 | 93 |
| Cl | 1,070 | 440 | 7 | 28 | 836 | 380 | 843 | 408 | 227 | 32 |
| + Acids (B) | +3,385 | +1,507 | +775 | +301 | +1,818 | +908 | +2,593 | +1,209 | +792 | +298 |
| Base balance (A − B) | −133 | −164 | −576 | −352 | +502 | +240 | −74 | −112 | −62 | −52 |
| Salt balance ½ (A + B) | | | | | | | | | 823 | 326 |

## COMMENT AND DISCUSSION

In our previous study [1] on milk modifications and the investigations there cited, as well as in all the mineral metabolism studies on breast-fed children quoted by Czerny and Keller [2] (table 264 A page 705), the intake was less than in the present study. The retention was also considerably smaller for each element, even though a much greater per-

2. Czerny, A. D., and Keller, A.: Des Kindes Ernährung: Ernährungsstörungen und Ernährungstherapie, Leipzig, 1923, vol. 1, p. 705.

centage of the intake was stored in the body. It is well known that the body will not permanently retain more of a single constituent than it requires for its normal development. Numerous experiments with metabolism of nitrogen indicate that an excess may be retained for a short period and later excreted. Data such as those given are rare in the literature, and more studies are urgently needed. Of special interest would be studies on breast-fed infants, on premature infants and on infants with rickets in the presence of an adequate supply of vitamin D. Increase in the rate of growth of experimental animals on improved diets has been so striking [3] that it becomes increasingly important to know whether the salt metabolism is a limiting factor and whether better growth can be attained by a more liberal supply of mineral elements in the food of infants.

Too far-reaching deductions should not be drawn from a single metabolism experiment. The conclusion that seems warranted from this study is that a baby on a skimmed milk diet, after the salt constituents are doubled, retains salts in approximately the same proportion, but in twice the amount.

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3. Osborne, T. B., and Mendel, Lafayette B.: The Relation of the Rate of Growth to Diet, I., J. Biol. Chem., **69**:661, 1926.

# THE INFLUENCE OF FEEDING ON CERTAIN ACIDS IN THE FECES OF INFANTS

## I. A COMPARISON OF THE EFFECTS OF BREAST MILK AND MODIFIED COW'S MILK ON THE EXCRETION OF VOLATILE ACIDS *

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From the time Pasteur [1] showed that volatile fatty acids could be the products of fermentation, the idea of relating them to intestinal peristalsis has interested medical men.[2] To pediatricians, however, these acids first assumed real importance during the years just preceding the Great War. At this time the high mortality of artificially fed children in the congested districts was a perplexing problem. Czerny sponsored the idea that volatile fatty acids developed by bacterial contamination of cow's milk were the etiologic factor. Against him were Finkelstein, Salge, Moro, Rietschel and Heubner, who believed other forces to be more important. During the years from 1911 to 1914 convincing evidence was offered in a series of excellent studies [3] stimulated apparently by Hans Bahrdt in connection with Huldschinsky, Stafford McLean

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* From the Otto Baer Fund for Clinical Research, the Sarah Morris Hospital for Children and the Nelson Morris Institute for Medical Research of the Michael Reese Hospital.

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1. Pasteur: Compt. rend. **46**:179, 1850; quoted by Bokai, Arch. f. exper. Path. u. Pharmakol. **24**:153, 1888.

2. Bokai, A.: Arch. f. exper. Path. u. Pharmakol. **24**:153, 1888. Von Braam, Houckgest: Pflüger's Arch. f. d. ges. Physiol. **5**:1872. Magnus: Pflüger's Arch. f. d. ges. Physiol. **102**:123, 1904. Katch and Boichers: Ztschr. f. exper. Path. u. Therap. **36**: 1913. Rona and Neukirch: Ibid. **146**:371, 1912. Fuhner: Ibid **102**:123, 1904. Trendelenburg: Ztschr. f. Biol. **61**:67, 1913.

3. Bahrdt, Edelstein, Langstein and Welde: Ztschr. f. Kinderh. **1**:139, 1911 Edelstein and Czonka: Ibid. **3**:313, 1912. Bahrdt and Bamberg: Ibid., p. 322 Bahrdt and Bamberg: Ibid. **4**:534, 1913. Huldschinsky, Kurt: Ibid., p. 366. Huldschinsky, Kurt: Ibid. **5**:475, 1913. Bahrdt and McLean: Ibid. **11**:143, 1914. Bahrdt and Edelstein. Ibid. **11**:403, 1914. Bahrdt, Edelstein, Hanssen and Welde: Ibid. **11**:416, 1914.

and others. These investigators, temporarily forgotten in the furor of the war, helped to clear the situation and did much to lay the scientific foundation for the further studies of that decade.

They devised a technic for determining small quantities of the volatile fatty acids. They noted the increased volatile fatty acid content of diarrheal stools. They compared quantitatively the volatile acids found in contaminated cow's milk, in the contents of the stomach and in diarrheal stools. They found much more in the stools than in the milk or in the stomach, which proved conclusively that the source of the acids was in the intestine. By experiment, they confirmed the previous observations as to the laxative effects of some of these acids, especially of acetic acid. They showed that not enough volatile fatty acid was formed in contaminated milk to cause diarrhea, while great quantities developed in the bowel.

These experiments, planned to answer the question of summer mortality, pointed the way to an even more interesting study; namely, a method of testing the basic theories of the Finkelstein school. Finkelstein,[4] after repeatedly calling attention to the harmless effects of diarrhea in the breast-fed infants, and to its deadly effects in the artificially fed, reasoned that there must be something in cow's milk which causes severe intestinal fermentation. In connection with Ludwig F. Meyer, he then described the clinical picture of alimentary intoxication, and, in the development of its treatment with protein milk,[5] suggested the theory that cow's milk while harmless in itself forms a medium unfavorable to the digestion and metabolism of the other elements of the milk.

The studies of Bahrdt and his associates gave us a method and stimulated us to undertake a more critical study of the Finkelstein hypothesis. Our investigations were planned as follows:

1. To determine whether there is any difference in the intestinal fermentation of carbohydrate as evidenced by the excretion of the volatile fatty acids in infants fed on breast milk and on appropriate mixtures of cow's milk.

2. To determine the effects on volatile fatty acid excretion of a considerable quantity of carbohydrate added to the two diets.

3. To attempt to determine the causative factors if differences existed.

This study concerns the first of these problems.

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4. Finkelstein: Personal conversations.

5. Finkelstein, H.: Jahrb. f. Kinderh. **65**:1; **66**:1, 1907; **68**:521, 1908; **71**:525, 1910; Text-Book of 1921. Finkelstein, H., and Meyer, L. F.: Ibid. **71**:525, 1910. Meyer, L. F.: Ibid. **65**:585, 1907.

## EXPERIMENTAL TECHNIC

We were unable to get any constant results with the technic used by the other investigators, namely, steam distillation in vacuum, and we finally turned to another method. Our procedure was as follows:

To facilitate the collection of specimens, the child, resting on a mattress in a bassinette, was placed on a suitably cut metabolic hammock. The mattress was made in four pieces with an opening for a small pail in the middle. The pail could easily be changed by the removal of one of the small pieces of mattress. Each specimen was collected in a separate pail and placed on ice immediately after defecation. The urine was collected in a separate receptacle. In a few instances, only one, but in the majority of cases, two twenty-four hour specimens taken twenty-four hours apart were collected each week.

At the end of each twenty-four hour period, the feces in each pail were weighed by difference and carefully rinsed with distilled water into a volumetric flask either 250 or 500 cc. in capacity. The mixture was made up to volume and filtered over night in the icebox. To 25 cc. of the filtrate was added 6 cc. each of 10 per cent sodium tungstate and two-thirds normal sulphuric acid to precipitate any proteins present which would interfere with the distillation. Twenty-five cubic centimeters of approximately tenth-normal phosphoric acid was then added. The mixture was made up to 120 cc. and filtered, and 110 cc. of the filtrate was used for the determination of volatile acids by the DuClaux method as modified by Gillespie and Walters.[6] All determinations were made in duplicate.

Most studies of the previous decades were isolated experiments on transient patients in the ward. It is self-evident that for purposes of comparison studies should be made on the same infant. Each subject was admitted to our wards immediately after birth and kept there until 6 months of age. Metabolism experiments were made once or twice a week. Feedings alternated between periods during which the infant was given breast milk and periods during which it was given appropriate modifications of cow's milk. The latter were changed when necessary to meet the requirements of growth.

## DIFFICULTIES

Since 1921 we have studied ten infants, and only four have gone through the six months' period without interruption of the experiment. To enumerate the difficulties and complications which can occur during an investigation of an infant in a hospital ward carried on for such a long period would fill a book. Two years' work was invalidated by imperfect chemical technic. We faced ruined experiments through otitis, measles and erysipelas. Wetnurses left at critical times or stayed and adulterated the breast milk. Ammonia system iceboxes failed at irregular intervals, and numerous specimens were hopelessly spoiled. Attending nurses got sick, lost specimens or permitted the urine and stool to mix. A most annoying complication, one which perplexed us for many months and which threatened to invalidate much of our work, was what we called "frame diarrhea." No matter what its feeding or how regular the stools, every time the baby was put on the metabolism frame it developed diarrhea. This diarrhea was unassociated with any

6. Gillespie, L. J., and Walters, E. H.: J. Am. Chem. Soc. **30**:2027, 1917.

subjective symptoms from the child, i.e., no influence on the weight curve or temperature, but was simply an increase in the number of bowel movements. The younger the baby the more susceptible he was to this complication (chart 1). We learned eventually that this was to some extent a nursing problem, and the difficulty was partially but not entirely overcome by having two special nursemaids for the child, one for the day and another for the night. In the majority of cases, however, placing the child on the frame increased the daily stool average by one or two.

## COMMENT

Our investigations embraced clinical considerations, such as temperature, weight curve, and the number and the weight of the stools.

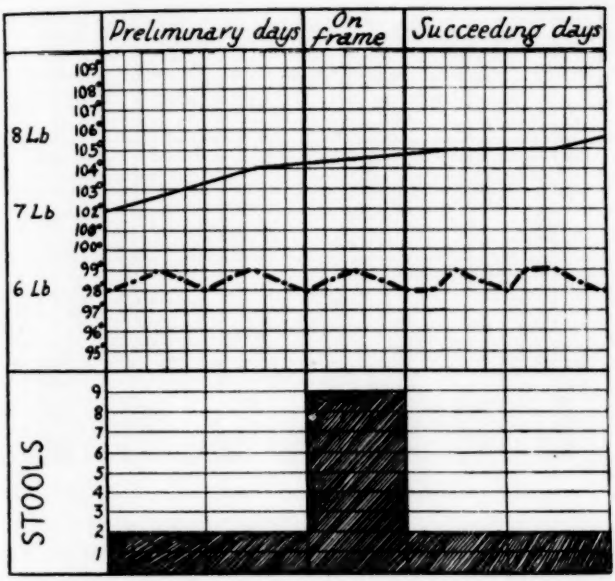


Chart 1.—The effect of the metabolism frame on the number of bowel movements.

Chemical studies considered the amount of acetic, formic and propionic acids, as well as the total volatile and total titratable acidity of the feces. In an effort to discover all possible relationships, we considered individual specimens as well as general averages for given periods on a certain diet. Calculations were made on the basis of the individual stool and on period averages; on the basis of per hundred grams of feces; per kilogram of body weight, and, finally, per hundred grams of feces per kilogram of body weight.

For simplification, the experimental results for one child, who in a general way showed reactions typical of all, have been graphically represented in chart 2. In tables 1, 2, 3 and 4, the results for all are tabulated.

At times great variations occurred in the acid output. These have not been included in the foregoing tables, but will be discussed in a later publication. It must be emphasized that the foregoing determinations are for children in a condition of clinical health; none of them include the results obtained when the child was suffering from any disturbance. A study of the tables and charts shows a number of striking differences between the two feedings.

*Weight of Stools.*—There is no question but that the average weight of the stools when the child was fed on cow's milk was greater than when it was fed on breast milk.

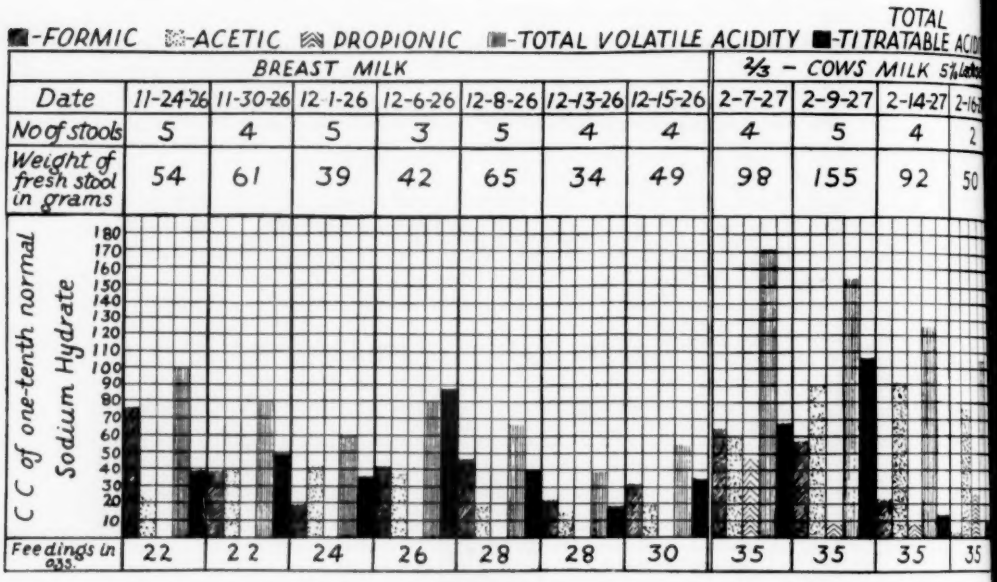


Chart 2.—The influence of breast milk and cow's milk mixtures on the quantity of feces and on the frequency of defecation, as well as on the excretion of volatile and total titratable acids.

*Total Titratable Acidity.*—When the child is fed on breast milk, the stools are usually acid, and the amount of free acid in a general way tends to be uniform. When the child is fed by bottle, this acidity shows great variability, often being close to the neutral. This corroborates the excellent studies of Brown and Tisdall,[7] and of Bosworth and his associates.[8]

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7. Tisdall and Brown: Acidity (hydrogen ion concentration) of Infants' Stools, Am. J. Dis. Child. **27**:312 (April) 1924; New York State J. Med. **25**:152, 1925.

8. Bosworth, Wilder, Blanchard, Brown and McCann: Studies of Infant Feeding; Bacteriologic Study of Feces and Food of Normal Babies Receiving Breast Milk, Am. J. Dis. Child. **23**:323 (April) 1922.

TABLE 1.—*Acid Excretion After Feeding*

| Number of Feedings | Quantity, Ounces | Number of Stools | Weight of Fresh Stool, Gm. | Formic | Acetic | Propionic | Total Volatile Acidity | Total Titratable Acidity |
|---|---|---|---|---|---|---|---|---|
| | | | **Breast Milk** | | | | | |
| 6 | 4 | 1 | 22.0 | 9.1 | 36.3 | .... | 31.4 | 20.8 |
| 6 | 4 | 1 | 26.0 | 0 | 28.9 | .... | 25.1 | 12.8 |
| 6 | 4½ | 1 | 14.2 | 0 | 29.2 | .... | 28.8 | 19.2 |
| 6 | 5 | 1 | 28.1 | 0 | 20.6 | .... | 22.8 | 9.9 |
| 6 | 5 | 2 | 41.9 | 7.5 | 61.5 | 0 | 44.5 | 21.2 |
| 6 | 5 | 2 | 24.4 | 9.2 | 22.6 | 0 | 21.6 | 22.7 |
| 6 | 5 | 4 | 57.7 | 39.2 | 68.1 | 0 | 72.1 | 84.6 |
| 6 | 5 | 4 | 24.7 | 29.2 | 37.4 | 0 | 44.1 | 158.3 |
| 6 | 5 | 2 | 56.4 | 47.7 | 79.2 | 0 | 85.0 | 72.4 |
| 6 | 5 | 1 | 50.2 | 27.0 | 39.2 | 0 | 44.1 | 29.6 |
| 6 | 5 | 1 | 11.5 | 11.9 | 11.4 | 0 | 15.2 | 10.0 |
| 6 | 5 | 6 | 93.0 | 11.0 | 81.5 | 0 | 65.1 | 42.3 |
| 6 | 5 | 2 | 45.1 | 16.3 | 56.8 | 0 | 50.4 | 38.3 |
| | | | **⅔ Cow's Milk, 6 per Cent Lactose** | | | | | |
| 6 | 5 | 9 | 47.1 | 0 | 49.3 | 22.6 | 54.5 | 6.4 |
| 6 | 5 | 2 | 29.7 | 0 | 43.9 | 9.3 | 39.5 | 7.2 |
| 6 | 5 | 3 | 74.9 | 0 | 51.4 | 17.0 | 51.4 | 4.9 |

The stools of this baby even when on breast milk were very constipated and full of anaerobic bacteria rather than bifidus.

TABLE 2.—*Acid Excretion After Feeding*

| Number of Feedings | Quantity, Ounces | Number of Stools | Weight of Fresh Stool, Gm. | Formic | Acetic | Propionic | Total Volatile Acidity | Total Titratable Acidity |
|---|---|---|---|---|---|---|---|---|
| | | | **Breast Milk** | | | | | |
| 6 | 5 | 1 | 16.1 | 12.5 | 32.3 | 0 | 44.8 | 29.6 |
| 6 | 5 | 1 | 24.4 | 20.2 | 42.1 | 0 | 62.3 | 65.4 |
| 6 | 5 | 1 | 18.6 | 19.0 | 26.0 | 0 | 45.0 | 47.5 |
| 6 | 5½ | 1 | 30.7 | 5.5 | 70.4 | 0 | 75.9 | 53.4 |
| | | | **½ Cow's Milk, 5 per Cent Lactose** | | | | | |
| 7 | 3½ | 7 | 91.9 | 0 | 148.9 | 6.7 | 155.6 | 86.5 |
| 7 | 4 | 3 | 46.6 | 38.6 | 37.8 | 8.9 | 85.8 | 39.3 |
| 7 | 4 | 3 | 23.5 | 3.4 | 24.8 | 5.2 | 33.4 | 2.5 |
| 7 | 4 | 4 | 29.8 | 21.6 | 27.1 | 8.8 | 57.5 | 12.0 |
| 7 | 4 | 4 | 42.0 | 50.3 | 3.1 | 23.8 | 77.2 | 10.7 |
| 7 | 4 | 2 | 37.3 | 0 | 36.5 | 25.5 | 62.0 | 28.5 |
| 7 | 4½ | 3 | 22.0 | 0 | 24.3 | 10.7 | 34.9 | 42.1 |
| 7 | 4½ | 1 | 10.4 | 0 | 14.4 | 3.5 | 17.9 | 39.0 |
| | | | **⅔ Cow's Milk, 6 per Cent Lactose** | | | | | |
| 6 | 5½ | 1 | 86.0 | 0 | 118.6 | 0 | 118.6 | 75.0 |
| 6 | 5½ | 6 | 118.1 | 82.6 | 103.3 | 0 | 185.9 | 100.0 |
| 6 | 5½ | 4 | 178.9 | 90.7 | 177.1 | 0 | 267.8 | 153.4 |
| 6 | 5½ | 4 | 102.7 | 27.9 | 92.1 | 26.8 | 146.8 | 28.3 |
| 6 | 5½ | 5 | 217.4 | 55.8 | 87.3 | 56.0 | 199.1 | 11.6 |

TABLE 3.—*Acid Excretion After Feeding*

| Number of Feedings | Quantity, Ounces | Number of Stools | Weight of Fresh Stool, Gm. | Formic | Acetic | Propionic | Total Volatile Acidity | Total Titratable Acidity |
|---|---|---|---|---|---|---|---|---|
| | | | **Breast Milk** | | | | | |
| 7 | 3 | 6 | 48.1 | 8.5 | 20.4 | 0 | 28.9 | .... |
| 7 | 3 | 6 | 51.2 | 26.0 | 29.3 | 0 | 55.3 | .... |
| 7 | 4 | 5 | 27.1 | 13.2 | 35.6 | 0 | 48.8 | .... |
| 7 | 4 | 11 | 87.6 | 52.9 | 13.5 | 0 | 66.4 | .... |
| 7 | 4 | 7 | 65.2 | 28.9 | 15.1 | 0 | 44.0 | .... |
| 7 | 4½ | 4 | 65.9 | 32.6 | 15.6 | 0 | 48.2 | .... |
| 7 | 4½ | 2 | 23.1 | 20.1 | 14.5 | 0 | 34.6 | .... |
| 7 | 4½ | 1 | 26.1 | 28.9 | 23.7 | 0 | 56.6 | .... |

*Volatile Acids.*—When the food consists of breast milk, the volatile acidity like the total titratability is in a general way uniform, and does not have any definite relation to the number or to the weight of the stools. When the infant is bottle-fed the volatile acid output is frequently greater than when the child is breast fed, and the quantity is more closely related to the number and to the weight of the stools. The last two factors seem to be more closely related when cow's milk is used than when breast feeding is employed.

As Bosworth [8] and his associates found, more formic acid forms when the infant is fed with breast milk, than when it is fed with cow's milk. Acetic acid tends to be higher in cow's milk.

In most cases an increase of formic acid is associated with an increase in total titratable acidity.

TABLE 4.—*Acid Excretion After Feeding*

| Number of Feedings | Quantity, Ounces | Number of Stools | Weight of Fresh Stool, Gm. | Formic | Acetic | Propionic | Total Volatile Acidity | Total Titratable Acidity |
|---|---|---|---|---|---|---|---|---|
| | | | Breast Milk | | | | | |
| 5 | 4½ | 5 | 54.5 | 26.1 | 23.9 | 0 | 100.0 | 39.4 |
| 5 | 5 | 4 | 61.1 | 39.0 | 40.6 | 0 | 79.6 | 49.0 |
| 5 | 5 | 5 | 38.9 | 17.9 | 42.4 | 0 | 60.2 | 34.6 |
| 5 | 5½ | 3 | 41.7 | 42.3 | 38.1 | 0 | 80.4 | 87.5 |
| 5 | 5½ | 5 | 64.9 | 46.2 | 19.4 | 0 | 65.6 | 40.0 |
| 5 | 5½ | 4 | 33.8 | 22.7 | 14.4 | 0 | 37.1 | 17.2 |
| 5 | 5½ | 4 | 49.3 | 32.2 | 19.4 | 0 | 51.6 | 33.9 |
| | | | ⅔ Cow's Milk, 5 per Cent Lactose | | | | | |
| 5 | 7 | 4 | 97.6 | 63.8 | 59.8 | 47.2 | 170.8 | 67.7 |
| 5 | 7 | 5 | 155.0 | 56.0 | 89.7 | 7.8 | 153.5 | 105.9 |
| 5 | 7 | 4 | 92.1 | 22.3 | 91.2 | 9.4 | 123.0 | 13.6 |
| 5 | 7 | 2 | 50.4 | 3.6 | 76.5 | 25.6 | 105.7 | 8.0 |
| 5 | 7 | 2 | 58.6 | 32.7 | 60.1 | 38.9 | 131.7 | 7.8 |
| 5 | 7 | 1 | 41.4 | 22.5 | 62.0 | 21.6 | 106.2 | 10.3 |

The striking difference between the results of the two kinds of feeding is that propionic acid appears in the stools of infants fed on cow's milk. To the best of our knowledge this has not been noted before. Almost without exception, propionic acid is an index of the stools of infants fed on cow's milk. It rarely appears in the stools of those fed on breast milk stool.

In studying these four infants, it is interesting to consider the influence of factors other than the diet.

*Effect of Age.*—There seems to be no definite relationship between the quantity of acids excreted and the age of the child. There may be a slight increase in a general way as the child gets older, but this is negligible compared with that caused by other factors. On cow's milk, there is an increase coincidental with the increase in the weight of the stool.

*Weight Curve.*—We could not find any relationship between the excretion of acid and the slight variations in the weight curve.

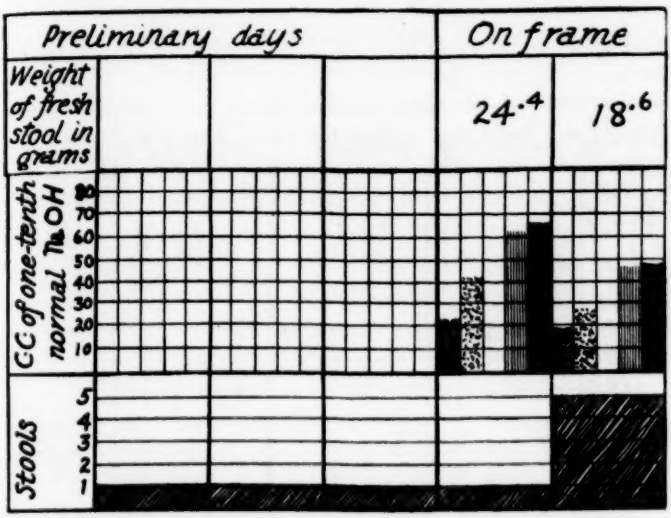


Chart 3.—The effect on the number of bowel movements and acid excretion when a breast-fed child is given six feedings of 5 ounces each and kept on the metabolism frame for two days.

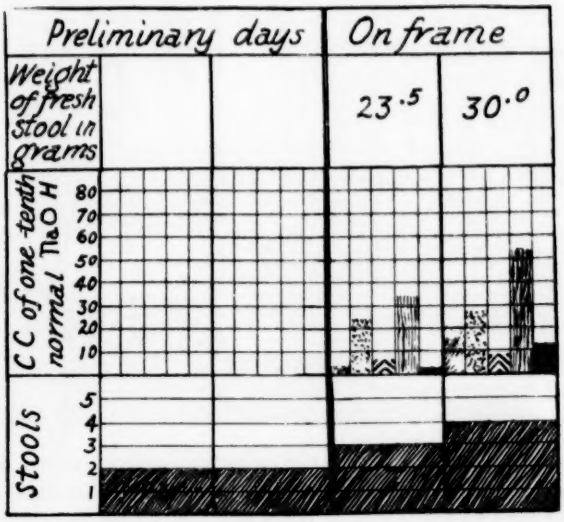


Chart 4.—The effect on the number of bowel movements and acid excretion when an artificially fed child is given seven feedings of 4 ounces each of a mixture of one half cow's milk and 5 per cent lactose and kept on the metabolism frame for two days.

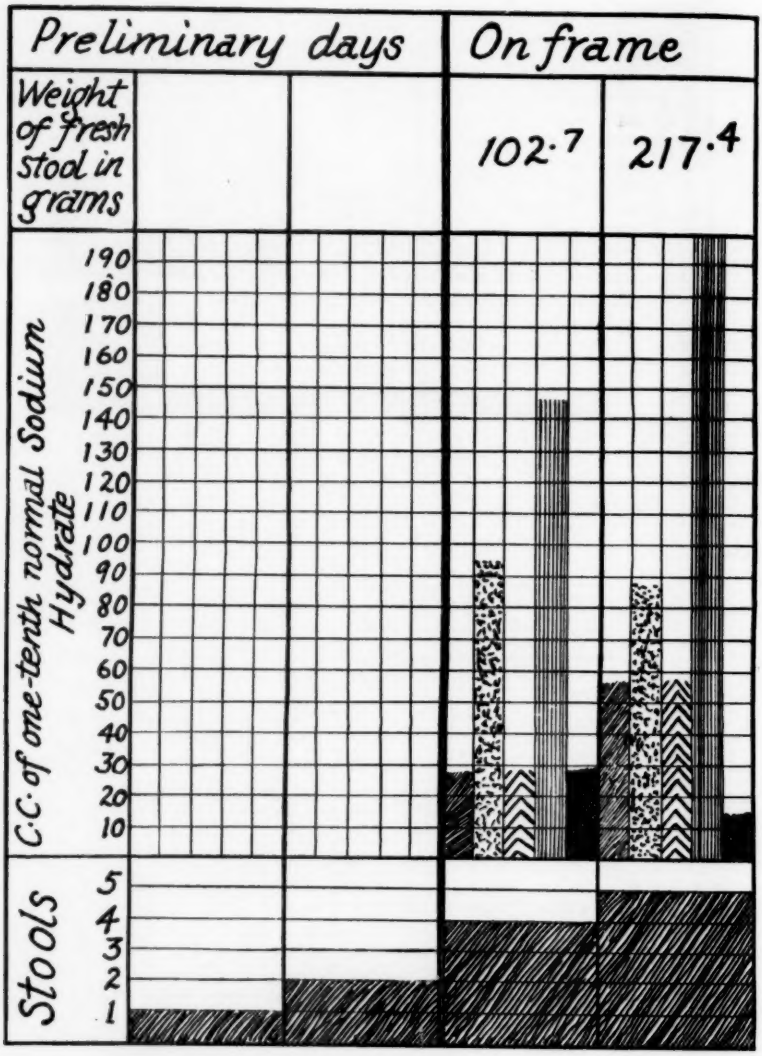


Chart 5.—The effect on the number of bowel movements and acid excretion when an artificially fed child with a tendency to diarrhea is given six feedings of 5½ ounces each of a mixture of two-thirds cow's milk and 6 per cent lactose and kept on the metabolism frame for two days.

*Influence of the Metabolism Frame.*—The most striking nondietetic influence was that of placing the child on the metabolism frame. Although he seemed absolutely comfortable and content, there was always, in spite of the most perfect nursing care, a slight increase in the number of stools on the day when the experiment was being made. To study this further, we conducted some experiments while the child was kept on the frame for two days. The results are shown in charts 3, 4 and 5. In most cases, the two day period did not increase excretion when the child was fed on breast milk. It did, however, when the child was fed on cow's milk, particularly when the infant was inclined toward diarrhea during the first day of the experiment.

*Number of Feedings.*—In a general way, the amount of acid was highest on the five feeding schedule and lowest on the seven feeding schedule, both when breast milk and when cow's milk was used. There were so many factors to be considered, however, and so few babies that this is only a superficial inference.

## CONCLUSIONS

In babies fed breast milk first and appropriate mixtures of cow's milk later, it was found that the volatile acid output and the total titratable acidity were relatively constant when breast milk was used and extremely variable when cow's milk mixtures were employed. The acidity of the stool of the infant fed on cow's milk was often much higher than that of the breast-fed infant; it was also frequently lower, showing an unstabilized condition in the intestinal tract.

It was also apparent that the stools of the infant fed on cow's milk were much heavier than those of the infant fed on breast milk, and that the acid excretion seemed to bear a closer relation to the weight of the stools of those receiving cow's milk than of those receiving breast milk.

While the study was primarily one of volatile acid excretion, one could not help observing the profound influence of factors other than food on the number of bowel movements. The placing of the child on the metabolism frame was a powerful agent in causing diarrhea.

Changing from breast milk to cow's milk almost invariably caused the appearance of propionic acid in the feces.

104 South Michigan Avenue.

# THE INFREQUENCY OF SEVERE RICKETS IN NEW ORLEANS AND VICINITY

## AN ATTEMPT TO CORRELATE SOME OF THE RESPONSIBLE FACTORS *

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NEW ORLEANS

The variation in the incidence and the severity of rickets for particular geographic locations is a long established fact. It is conceded generally among clinicians of this country that the incidence of severe rickets in the extreme Southern states is much less than in the Northern states. Patients with rickets showing marked deformity of the bones are extremely rare in the clinics of New Orleans. Mild deformities of the bones, such as eversion of the ribs at the infracostal margins, beading at the costochondral junctions, epiphyseal enlargements at the wrists, flattening of the walls of the chest and first degree bowing of the legs are fairly common here, but these conditions are rarely encountered in their advanced stages. The usual manifestations in the cases of rickets are: Moderate enlargement of the epiphyses at the wrists, beading at the costochondral junctions, slight deformities of the chest, parietal bosses, delayed dentition, muscular weakness and sweating of the head. Only occasionally are well marked "rachitic rosary," or a "pigeon breast" found. Lateral flattening of the walls of the chest to the extent of severely impinging on the excursions of the diaphragm, or extreme deformity of the long bones are so rare that they are clinical novelties. The patients with more than a fairly well marked degree of rickets among those coming to the clinic nearly always give a history of extreme faulty feeding, with hardly any general care. At the children's division of the outpatient service, Touro Infirmary, there have been more than 2,000 new admissions during the past year and approximately 13,000 additional patients have been treated within that time, with only one case of rickets which showed severe deformity of the long bones. In order to gain some estimate as to the incidence of rickets among patients coming to the clinic, the records of 300 children, aged 3 years or under, were taken from the files at random for study. A diagnosis of rickets was made on three or more positive rachitic symptoms in a patient, such as beading of the ribs, enlarged epiphyses, delayed dentition,

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* From the Department of Pediatrics, Tulane University of Louisiana School of Medicine.

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muscular weakness and bow legs. The result of this study showed that 45 per cent of these children had rickets. The disease is therefore common in this community, but it does not seem to advance nearly as far here as it does in certain other localities of the country.

In view of the incomplete knowledge concerning the etiology of rickets, the mild form of the disease as found in this locality cannot be fully explained at present. New Orleans certainly has its share of the poorer classes, in both the colored and the white races; therefore, this factor could not be considered when I attempted a solution. In the light of the knowledge concerning the influence of sunlight on rickets, one might expect to find at least a partial explanation in the geographic location. The climate and the fertility of the soil in this section of the country insure a bounteous supply of fresh green vegetables the year round, which makes this type of food available to all classes of the population. Experimental evidence indicates that green vegetables possess some antirachitic properties and exert a pronounced influence on both growth and development, which, if true, would place this item in the category of ameliorating the disease. The average annual temperature is mild, permitting children to play outdoors at all times during the year, and it is probable that as a result of this they get a greater percentage of the possible sunshine than children do in sections farther north, where they are kept in close confines during the rigorous winter months. Also, the clothing of children of this vicinity is scant and of a much lighter texture than that worn by children in regions to the north. New Orleans has an area of 196.25 square miles with a population of 387,019, according to the census of 1920. The buildings as a whole are of low construction and are separated from each other in the residential districts by unusually large lots or open spaces, which afford almost the maximum opportunity for available sunlight.

It occurred to me that with these peculiarities extant in a particular section of the South, it might be worth while to investigate, so far as possible, some of the influences already mentioned in their relations to a group of children coming to the clinic, with the object in view of trying to find out what factors are responsible for the mild form of rickets in New Orleans.

## REVIEW OF THE LITERATURE

There is no record that tells when rickets was first recognized as a disease entity, but Glisson [1] definitely described the condition in 1650. There are fairly accurate clinical composia to be found in the literature on rickets as far back as 1630. Dating from the description of the

1. Glisson, F.: De Rachitide, ed. 2, London, 1660.

disease by Glisson, volumes have been written on its various phases, and many brilliant inferences have been drawn as to its etiology, prophylaxis and treatment. Credit for the full development of the present sum of knowledge of rickets does not belong to any one person, but is shared by many.

The earlier authors on rickets in this country were almost unanimously agreed that the disease was a condition found only in the Old World. In 1868, Bauer [2] of New York, stated that rickets was the rarest malady on the western continent. At that time statistics were not lacking concerning the incidence of the disease in the countries of Europe. In 1871, Ritchie [3] found that the incidence of rickets among the children coming under his care at the Hulme Dispensary, Manchester, England, was about 30 per cent. In the reports of St. Bartholomew's Hospital, London, for 1872, Gee [4] stated that the incidence of rickets in children under 2 years was 30.3 per cent. There was much discussion in this country at that time as to whether the disease was present here to any considerable extent. In 1872, Parry [5] took the stand that the frequency of rickets in this country had been much underestimated and reported for that year that 28 per cent of all children at the Philadelphia Hospital had rickets. He added further: "This estimate is rather under than beyond the truth." In 1895, Snow,[6] in the reports of the Buffalo Fresh Air Mission Hospital, placed the incidence of rickets among Italian children under 3 years of age at 70 per cent and from 11 to 12 per cent among other nationalities. After a clinical survey in Boston and vicinity in 1900, Morse [7] reported rickets in 80 per cent of children under 2 years of age among the poorer classes. Endemicity of the disease, therefore, ceased to be a question many years ago, and the query now is: "How prevalent is it?"

After studying the results of the various clinical surveys which have been made in different sections of the country relative to the incidence of rickets, I found that the estimates range from 25 per cent in Denver (1925), according to Forbes and Green,[8] to 80 per cent in Boston

2. Bauer, H. L.: Lectures in Orthopaedic Surgery, New York, 1868, p. 213.

3. Ritchie, C. C.: Frequency of Rickets at the Hulme Dispensary, Manchester (England), M. Times & Gaz., Jan. 7, 1872.

4. Gee, F. H.: St. Bartholomew's Hospital Reports, London **4**:70, 1872.

5. Parry, J. S.: Observation on the Frequency and Symptoms of Rickets, with Results of the Author's Clinical Experience, Am. J. M. Sc. (new series) **63** and **64**:17, 1872.

6. Snow, I. M.: Incidence of Rickets in Buffalo, Tr. Am. Pediat. Soc. **12**: 18, 1895.

7. Morse, J. L.: Frequency of Rickets in Boston and Vicinity, J. A. M. A. **34**:724 (March 24) 1900.

8. Forbes, R., and Green, B.: Incidence of Rickets in Colorado, with Report of Clinical Survey and Climatological Observation, Colorado M. J. **22**:69, 1925.

(1900), according to Morse.[7] A tenable question would be: Why are there such wide variations in incidence for the different localities? Different criteria have no doubt been used in arriving at a diagnosis by the different groups of workers, but this by itself could not account for such variations in incidence.

Those who have written on the geographic distribution of rickets say that it is almost unknown in the Far North among the Eskimos and the Scandinavian people; that it is rare in the tropics and at high altitudes; rare, except in the larger cities, in the countries of Southern Italy, Spain, Egypt, Syria, China and Japan.[9] Many writers of the past have pointed out that it is primarily an urban disease and that it is relatively infrequent among children of rural districts.

Until less than a decade ago, physicians were in complete darkness concerning the etiology of rickets. In 1919, Mellanby[10] proved that it could be produced, modified, or prevented in animals by diet. He produced rickets in puppies by feeding them on a diet consisting of minimal amounts of skimmed milk, an unlimited amount of bread, orange juice, yeast and salt. To modify or to prevent the disease, he added to this diet the different oils, meat and fats. Another group of workers, including Findlay and Paton,[11] were unwilling to accept Mellanby's work as conclusive, believing that environment was the most potent factor in the etiology. They repeated Mellanby's work, but instead of keeping all the animals in laboratory cages in the city, as Mellanby had done, they kept one group in the city and another group in the country. The animals kept in the city developed rickets, while those kept in the country did not. This controversy contributed much to the present belief that both diet and sunlight are responsible factors in the etiology of rickets. The work of Mellanby and his opponents is probably the most important milestone in the developmental history of rickets, because it afforded a tangible basis for further investigations. Shortly after this work, both clinical and laboratory investigations followed in rapid succession, so that today if one attempts to review the literature which has accumulated on rickets within the past six or eight years, he is astounded at its volume and is forced to conclude that much of the story has already been told.

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9. Dick, J. L.: A Study of Economic Conditions and Their Effects on the Health of the Nation, London, 1922. Findlay, L.: Rickets: A Historical Note, Glasgow M. J. **91**:147, 1919. Palm, T. A.: The Geographical Distribution and Aetiology of Rickets, Practitioner **65**:270, 1890.

10. Mellanby, E.: An Experimental Investigation in Rickets, Lancet **1**:470, 1919.

11. Paton, D. N.; Findlay, L., and Watson, A.: An Experimental Study in Rickets, Brit. M. J. **2**:625 (Dec. 7) 1919.

Hess,[12] Steenbock,[13] Park [14] and others showed that sunlight stimulates the concentration and retention of calcium and phosphorus in the blood. Maynard, Goldberg and Miller [15] (1925) showed that the ash content of swine is greater when exposed to sunlight in the spring than when exposed to sunlight in the winter. Orr, MaGee and Henderson [16] (1924) found that the calcium and phosphorus retention in the serum of pigs was increased by exposure to a carbon arc lamp for one hour a day for twenty-four days. Kramer, Casparis and Howland [17] (1922) treated a number of definitely rachitic children by irradiation with the mercury vapor quartz lamp and found that the disease promptly healed, with a gradual rise in the inorganic phosphorus of the blood from a low level (2.7 to 3.2 mg. per cent) to about 6 mg. per cent. Hughes, Nitcher and Titus [18] (1925) found that chickens failed to develop rickets on the usual rickets-producing diet, if irradiated with the mercury vapor quartz lamp for ten minutes twice a day for eight weeks.

Howland and Kramer [19] (1922) found that all children with less than 3 mg. per cent of inorganic phosphorus in the blood showed evidence of rickets and pointed out that the ratio which exists between the calcium and the phosphorus in the blood of nonrachitic children is practically identical with the ratio of these elements in tertiary calcium phosphate, the salt which forms from 85 to 90 per cent of the mineral content of bone. They contend that it is usually the phosphorus deficiency in rickets which prevents the normal deposition of calcium in the bones. They offer as evidence supporting this theory the fact that the blood of children with active rickets may show a normal calcium

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12. Hess, A. F.: Prevention and Cure of Rickets by Sunlight, Am. J. Pub. Health **22**:104 (Feb.) 1922. Hess, A. F.; Unger, L. J., and Pappenheimer, A. M.: A Further Report on Prevention of Rickets by Light Rays, Proc. Soc. Exper. Biol. & Med. **9**:238, 1921-1922; Experimental Rickets in Rats: VII. The Prevention of Rickets by Sunlight; by the Rays of the Mercury Vapor Lamp and by the Carbon Arc Lamp, J. Exper. Med. **36**:427 (Oct.) 1922.

13. Steenbock, M.: Nutritional Requirements of Baby Chicks; Relation of Light to Growth of Chickens, J. Biol. Chem. **58**:34 (Nov.) 1923.

14. Park, E. A.: Etiology of Rickets, Physiol. Rev. **3**:106, 1923.

15. Maynard, L. A.; Goldberg, S. A., and Miller, R. C.: Influence of Sunlight on Bone Development of Swine, J. Biol. Chem. **45**:645 (Oct.) 1925.

16. Orr, J. B.; MaGee, H. E., and Henderson, J. M.: Influence of Sunlight on the Calcium and Inorganic Phosphorus in the Serum of Pigs, Am. J. Physiol. vol. 59, proc. 25, 1924.

17. Kramer, B.; Casparis, H., and Howland, J.: Ultraviolet Radiation in Rickets. Effects on the Calcium and Inorganic Phosphorus Concentration in the Serum, Am. J. Dis. Child. **24**:20 (July) 1922.

18. Hughes, J. S.; Nitcher, C., and Titus, R. W.: The Relative Value of Ultra-Violet Light and Irradiated Air in Preventing Rickets in Chickens, J. Biol. Chem. **63**:205 (March) 1925.

19. Howland, J., and Kramer, B.: Factors which Determine the Concentration in the Serum of Rats, Bull. Johns Hopkins Hosp. **33**:313, 1922.

reading, while in all such cases the inorganic phosphorus is diminished. As a result of this work, physicians have come to think of rickets as a condition in which there may be a deficiency in either calcium or phosphorus, or a deficiency in both (the calcium-phosphorus product criterion).

Hess and Lundagen [20] (1922) showed that there is a seasonal tide in the inorganic phosphorus content of the blood of children, the lowest ebb of the phosphorus tide occurring about March and the highest peak of the tide about June. Hess and Unger [21] (1922) pointed out the marked similarity between the seasonal phosphorus curve and the curve of clinical rickets and called attention to the fact that the peak of the rickets curve is coincident with the ebb of the phosphorus curve and vice versa.

Hess and Unger [22] (1922) found in New York City that rickets in breast-fed negro children was invariably associated with a deficiency of green vegetables in the diets of the mothers. Hess and Weinstock [23] (1925) demonstrated that certain food substances which ordinarily did not possess protective properties against rickets acquired this property to a remarkable degree after irradiation with the mercury vapor quartz lamp. After irradiation, cottonseed oil and linseed oil, in 0.1 cc. doses, were found to protect rats against rickets. Green wheat, fresh lettuce leaves and refined wheat flour were all found to take on this antirachitic property after irradiation. Hess, Weinstock and Helman [24] (1925) extended the study of irradiated substances further and showed that even chemical substances, like cholesterol and phytosterol, take on the antirachitic property after irradiation. In connection with this work, they have formulated a most interesting hypothesis, bearing on the pathogenesis of rickets. They suggested that the cholesterol of the deeper layers of the epidermis is activated or converted by the solar rays, so as to protect against rickets. But they subtend this hypothesis by the fact that neither cod liver oil nor its concentrated extract will protect against rickets when it is injected subcutaneously into rachitic animals. Kramer [25] (1925) treated children with active rickets by

20. Hess, A. F., and Lundagen, M. A.: Seasonal Tide in Blood Phosphates in Infants, J. A. M. A. **79**:2210 (Dec. 30) 1922.

21. Hess, A. F., and Unger, L. J.: Interpretation of the Seasonal Variation in Rickets, Am. J. Dis. Child. **22**:186 (Aug.) 1921.

22. Hess, A. F., and Unger, L. J.: The Diet of the Negro Mother in New York City, J. A. M. A. **70**:900 (March 30) 1918.

23. Hess, A. F., and Weinstock, M.: A Further Report on Imparting Anti-Rachitic Properties to Inert Substances by Ultra-Violet Irradiation, J. Biol. Chem. **63**:297 (March) 1925.

24. Hess, A. F.; Weinstock, M., and Helman, F. D.: The Antirachitic Value of Irradiated Cholesterol and Phytosterol, J. Biol. Chem. **63**:305 (March) 1925.

25. Kramer, B.: Rickets in Infants. Treatment with Irradiated Milk. Am. J. Dis. Child. **30**:195 (Aug.) 1925.

giving them irradiated cow's milk, orange juice and a cereal, with the results of complete healing of the disease and a return to the normal level of the inorganic phosphorus of the blood within four weeks. Two other rachitic children fed in the same way, except that nonirradiated cow's milk was given, did not show signs of improvement.

Howland and Kramer [19] (1922) demonstrated that the addition of large amounts of calcium and phosphorus to diets normal in these constituents did not cause an increase in the calcium and phosphorus in the blood of children. But in the case of children subsisting on diets deficient in both calcium and phosphorus, they were able to increase the concentration of these elements in the serum by the addition of cod liver oil. Greenwald [26] (1925) proved that by the subcutaneous injection of calcium and phosphorus these elements are temporarily increased in the serum, but he found that this increase is fleeting and that the tendency is for the calcium and phosphorus to return quickly to their normal levels.

Collip and his co-workers [27] (1925) showed that the normal calcium level in the serum of dogs is controlled by a hormone of the parathyroid glands. They studied both normal and parathyroidectomized dogs by injecting them with an aqueous extract of parathyroid glands from oxen. They observed that the dogs with parathyroid tetany could be restored by injecting this extract, or thrown back into the condition of parathyroid tetany by withholding it. The parathyroid extract raised the blood calcium in the normal dogs to enormous levels (more than 20 mg. per cent within less than thirty-six hours), larger doses producing the syndrome of hypercalcemia and death. They administered the parathyroid extract to a number of human subjects and found that the serum calcium levels were increased. The blood serum phosphorus was not materially disturbed in the early stages of hypercalcemia, but it rose to high levels during the terminal stages.

Working in California, Risenfeld, Handelman and Rose [28] (1925) concluded that precipitation influences the inorganic phosphorus content of the blood of newly born children more markedly than does sunlight.

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26. Greenwald, I.: Excretion of Calcium, Phosphorus and Magnesium, After Injection of Calcium Chloride, Sodium Phosphate, or Both, J. Biol. Chem. **76**: 201 (Nov.) 1925.

27. Collip, J. B.: The Extract of A Parathyroid Hormone Which Will Prevent or Control Parathyroid Tetany and Which Regulates the Level of Blood Calcium, J. Biol. Chem. **63**:395 (March) 1925. Collip, J. B.; Clark, E. P., and Scott, J. W.: The Effect of A Parathyroid Hormone on Normal Animals, J. Biol. Chem. **63**:439 (March) 1925.

28. Risenfeld, E. A.; Handelman, I. and Rose, A. R.: Inorganic Phosphorus in the Blood of the Newly Born: Its Seasonal Variation and Relation to Rickets, Am. J. Dis. Child. **30**:646 (Nov.) 1925.

They offered in explanation of their observation the physical principle, that the moisture washes the dust particles from the air, thus affording a more penetrable ultraviolet ray from the sun.

## METHOD

The work was planned to cover a period of one year, extending from November, 1925, to November, 1926. One hundred children were taken at random on admission to the outpatient service, Touro Infirmary, New Orleans. A diagnosis of the outstanding condition was made soon after admission, with particular attention to coexisting signs of rickets. All children, aged 1 year or less, were given cod liver oil and sun baths as a routine, in so far as these could be governed in dealing with patients in the clinic. The group of 100 consisted of thirty-four white and sixty-six colored children, the ages ranging from 1 month to 11 years. There were seventy-four nursing infants of 1 year or less, the remainder being made up of age groups as follows: 2 years, eight; 3 years, two; 4 years, one; 5 years, one; 6 years, one; 7 years, two; 8 years, three; 9 years, three; 10 years, two, and 11 years, three. Forty-eight of the seventy-four nursing infants were breast fed altogether; five were breast fed and bottle fed and twenty-one were bottle fed.

The points considered in relation to the individual members of the group were: (1) the presence or absence of rickets; (2) monthly determinations of the inorganic phosphorus in the blood of each patient; (3) single determinations of calcium soon after admission; (4) the percentage of possible sunshine for each month; (5) the mean of the average daily temperature for each month; (6) the amount of precipitation in inches for each month; (7) the quality of the diets of the nursing mothers and of the older children, with particular reference to the amount of green vegetables in these diets; (8) the presence or absence of disease in members of the group which might influence the concentration and retention of calcium and phosphorus in the blood, and (9) environment, with reference to available sunlight, sanitation and hygiene.

A diagnosis of rickets was made or disproved by collaborating the physical manifestations with roentgen-ray examinations of the bones and chemical analyses of the blood. In two or three instances, in which the physical manifestations were at variance with the other diagnostic aids, precedence was given the physical manifestations.

The meteorologic data were obtained from the monthly summaries of the reports by the U. S. Department of Agriculture.

Trained social workers visited the homes for the purpose of collecting certain data. A protocol for the guidance of the social worker was provided, which when filled out by her, gave complete information as

to housing conditions, playground facilities, available sunlight, home environment and hygiene and general care. The diets of the nursing mothers and those of the older children were tabulated monthly, by discussing them with the mothers at subsequent visits.

Blood was collected by puncture of the longitudinal sinus, or by venipuncture and the inorganic phosphorus and calcium contents determined shortly afterward. The method used for the determination of phosphorus was Brigg's modification of the Bell-Doisy phosphate method;[29] that used for the determination of calcium was Tisdall's modification of the Kramer-Tisdall method.[30]

Monthly ranges of the inorganic phosphorus content of the blood for the entire group have been graphically represented in the form

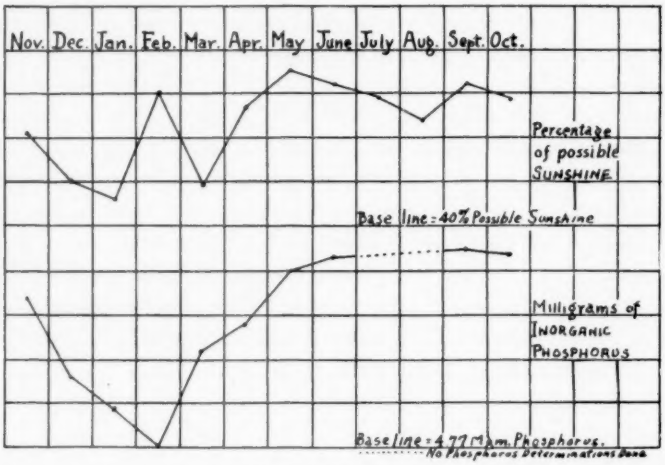
Nov. Dec. Jan. Feb. Mar. Apr. May June July Aug. Sept. Oct.
Percentage of possible SUNSHINE
Base line = 40% Possible Sunshine
Milligrams of INORGANIC PHOSPHORUS
Baseline = 4.77 Mgm. Phosphorus.
No Phosphorus Determinations Done

Chart 1.—The effect of sunshine on the content of inorganic phosphorus in the blood of children. It should be noted that when the inorganic phosphorus is below its maximum level, a rise or a decline in the amount of sunshine for a given month is followed in direct proportion the succeeding month by a rise or a decline, as the case may be, in the content of inorganic phosphorus in the blood. In the phosphorus curve of all three charts, one of the squares equals 0.2 mg. In the curve representing the amount of possible sunshine, one square equals 10 per cent.

of curves (charts 1, 2 and 3). The phosphorus curve has been plotted against the percentage of possible sunshine for each month, the mean of the average daily temperature for each month and the amount of precipitation in inches for each month. Also a comparative climatologic

29. Briggs, A. P.: A Modification of the Bell-Doisy Phosphate Method, J. Biol. Chem. **53**:13 (July) 1922.

30. Tisdall, F. F.: Kramer-Tisdall Method for Determination of Calcium in Serum: Tisdall's Modification, J. Biochem. **56**:459, 1923.

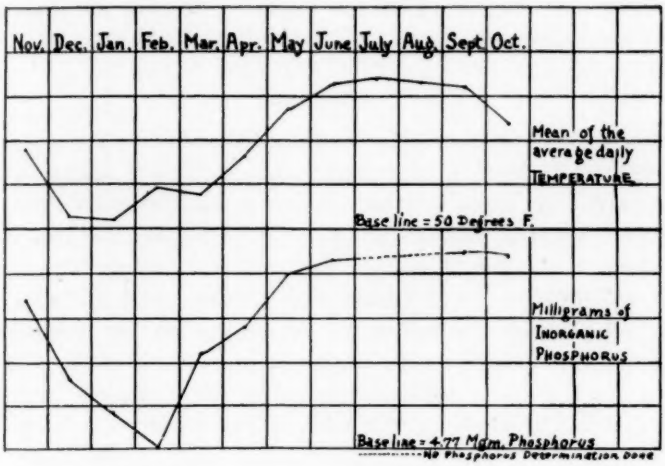
Nov. Dec. Jan. Feb. Mar. Apr. May June July Aug. Sept. Oct.
Mean of the average daily TEMPERATURE
Base line = 50 Degrees F.
Milligrams of INORGANIC PHOSPHORUS
Base line = 4.77 Mgm. Phosphorus
No Phosphorus Determination Done

Chart 2.—The monthly range of the average daily temperature as compared to the monthly range of the content of inorganic phosphorus in the blood of the children of the group. In the curve representing the mean of the average daily temperature, one square equals 10 degrees F.

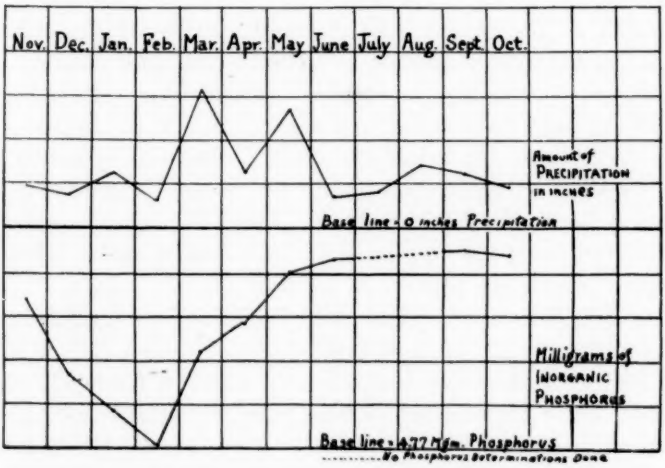
Nov. Dec. Jan. Feb. Mar. Apr. May June July Aug. Sept. Oct.
Amount of PRECIPITATION IN INCHES
Base line = 0 inches Precipitation
Milligrams of INORGANIC PHOSPHORUS
Base line = 4.77 Mgm. Phosphorus
No Phosphorus Determinations Done

Chart 3.—The absence of any relation between the amount of rainfall and the content of inorganic phosphorus in the blood of the children of the group. In the precipitation curve, one square equals 5 inches.

table has been arranged (table 3) between New Orleans, New York, Denver and Los Angeles, which is designed to show the influence of certain meteorologic phenomena on the incidence and severity of rickets in these localities.

## RESULTS

In twenty-six patients, active and partially healed rickets was found on admission; in sixteen, the disease was definitely active, while ten showed partial healing. Of the cases of active rickets, three occurred among the breast fed, twelve among the bottle fed and one case occurred in an older child, aged 3 years. Ten or twelve of the older children showed definite evidence of healed rickets. One child in the group had tetany, and showed a considerable diminution in the serum calcium (7.4 mg. per cent), but a normal inorganic phosphorus reading (5.55 mg. per cent). Not any others of the group were thought to have any disease that might influence the calcium and phosphorus levels of the blood.

Every child with active rickets responded more or less promptly to treatment with cod liver oil, regulation of diet and sun baths. A rise in the inorganic phosphorus content of the blood was the earliest sign of improvement, being noticeable in most cases within one month after treatment was instituted.

Table 1 shows that there was a seasonal variation in the content of inorganic phosphorus in the blood. It will be noted also that the ebb of the phosphorus tide began in November, reached its lowest level in February and began to rise gradually in March, reaching its summer level in May. The lowest total monthly average of phosphorus was 4.77 mg. per cent for February, and the highest total monthly average was 5.73 mg. per cent for September. There were only slight fluctuations in the total phosphorus levels from May until December. The ages of the children studied ranged from 1 month to 11 years, but this apparently did not make any difference in the content of inorganic phosphorus in the blood. The serum phosphorus levels in some of the older children were even higher than they were in quite a few of the nonrachitic nursing infants.

Table 2 indicates the status of the calcium and phosphorus levels at the time of admission and also the calcium-phosphorus products. It will be noted that only a few of the calcium readings fall below the normal limits for children, while the inorganic phosphorus was low in every case of well defined rickets. The average for the calcium-phosphorus products of this series is high, as compared to the results of other investigators of the North.

Green vegetables, such as spinach, turnips, carrots and cabbage, featured prominently in the diets of the nursing mothers. This was

TABLE 1.—*Monthly Determinations of the Inorganic Phosphorus in the Blood of Children* *

| Case | 1925 and 1926 | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| | Nov. | Dec. | Jan. | Feb. | March | April | May | June | Sept. | Oct. |
| 1 | 6.25 | 5.00 | 7.00 | 5.00 | 6.65 | 6.25 | 7.65 | .... | .... | 5.65 |
| 2 | 6.25 | 5.00 | 5.90 | 4.15 | 3.35 | 5.35 | 5.85 | | | |
| 3 | 3.65 | 4.55 | .... | 4.25 | 4.13 | 4.55 | 5.00 | | | |
| 4 | 7.65 | 6.65 | 5.55 | 5.00 | 5.60 | 5.60 | 5.00 | .... | .... | 6.15 |
| 5 | 5.90 | 6.25 | 5.00 | 4.35 | 5.00 | .... | 5.35 | | | |
| 6 | 4.55 | 5.00 | 5.25 | 4.75 | 5.55 | 5.00 | 5.25 | | | |
| 7 | 5.90 | 6.65 | 4.55 | 4.25 | 5.00 | 5.00 | 5.85 | | | |
| 8 | 7.10 | 5.25 | 5.00 | 4.55 | 5.00 | 5.35 | Died | | | |
| 9 | 6.25 | 5.00 | 4.00 | 3.45 | 4.10 | 5.75 | 5.35 | | | |
| 10 | 6.65 | 7.10 | 5.25 | 5.00 | 5.75 | 6.35 | 7.10 | | | |
| 11 | 8.30 | 6.25 | 5.55 | 4.25 | 4.75 | 5.15 | 5.00 | .... | .... | 6.25 |
| 12 | 8.30 | 7.65 | 6.25 | 5.55 | 5.25 | 6.04 | 7.05 | | | |
| 13 | 3.03 | 4.55 | .... | 3.00 | 4.55 | 5.00 | 5.35 | .... | .... | 3.70 |
| 14 | 4.00 | 5.00 | .... | 4.75 | 5.45 | 5.00 | 5.25 | | | |
| 15 | 2.30 | 4.50 | 4.35 | 5.00 | 4.75 | 4.70 | 5.00 | .... | .... | 5.00 |
| 16 | 5.00 | 6.25 | 7.65 | 5.55 | 5.85 | 5.55 | 6.15 | | | |
| 17 | .... | 7.65 | 5.00 | 6.65 | 4.25 | 4.00 | 4.75 | | | |
| 18 | .... | 7.10 | 6.25 | 5.25 | 7.00 | 6.65 | 5.85 | .... | .... | 6.25 |
| 19 | .... | 7.10 | .... | 5.55 | 5.75 | 5.35 | 6.15 | | | |
| 20 | .... | 5.00 | 6.65 | 4.15 | 5.85 | 6.65 | 5.75 | | | |
| 21 | .... | 4.50 | 3.03 | 4.00 | 4.00 | 4.34 | 5.00 | | | |
| 22 | .... | 6.65 | 5.55 | 6.25 | 7.10 | 5.55 | 5.00 | | | |
| 23 | .... | 7.10 | 6.25 | 5.00 | 6.65 | .... | 5.85 | | | |
| 24 | .... | 8.65 | 8.15 | 5.00 | 5.25 | 5.55 | 6.65 | | | |
| 25 | .... | 6.25 | 7.10 | 4.55 | 4.75 | 6.65 | 5.25 | .... | .... | 7.65 |
| 26 | .... | 5.00 | 6.65 | 4.15 | 6.25 | 5.85 | 5.00 | 5.25 | .... | 5.00 |
| 27 | .... | 5.25 | 5.90 | 4.55 | 5.00 | 5.15 | 5.75 | 5.85 | | |
| 28 | .... | 7.10 | 7.65 | 6.65 | 6.04 | 5.75 | 6.65 | | | |
| 29 | .... | 4.55 | 5.50 | .... | 4.25 | 5.00 | 5.55 | 5.85 | | |
| 30 | .... | 4.00 | 6.25 | 5.55 | 6.65 | 5.85 | 6.65 | | | |
| 31 | .... | 5.55 | 4.35 | 3.08 | .... | 5.25 | 5.00 | | | |
| 32 | .... | 6.65 | 7.10 | 5.20 | 5.00 | 5.00 | 5.55 | .... | .... | 6.65 |
| 33 | .... | 8.15 | 5.25 | 3.85 | 4.75 | 4.25 | 5.25 | .... | .... | 6.15 |
| 34 | .... | 6.25 | 5.55 | 4.25 | 5.00 | 4.15 | 6.04 | | | |
| 35 | .... | 4.55 | 5.00 | 4.10 | 4.80 | 4.80 | 5.25 | | | |
| 36 | .... | 5.00 | 4.35 | 4.00 | 5.00 | 3.00 | 4.25 | | | |
| 37 | .... | 5.00 | 4.35 | 5.00 | 5.00 | 5.25 | 5.00 | .... | .... | 5.85 |
| 38 | .... | .... | 4.30 | 5.00 | 4.75 | 5.00 | 5.25 | | | |
| 39 | .... | .... | 4.15 | 4.75 | 4.25 | .... | 5.55 | | | |
| 40 | .... | .... | 4.55 | 4.90 | 5.75 | 5.85 | 6.65 | | | |
| 41 | .... | .... | 5.55 | 4.85 | 5.00 | 5.00 | 5.75 | | | |
| 42 | .... | .... | 6.65 | 5.55 | 6.15 | 7.10 | 6.15 | .... | 7.10 | |
| 43 | .... | .... | 4.35 | 6.65 | 5.25 | 6.15 | 5.75 | .... | Died | |
| 44 | .... | .... | 4.65 | 3.03 | 4.70 | 5.35 | 5.00 | | | |
| 45 | .... | .... | 4.35 | 4.15 | 4.80 | 4.70 | 5.00 | .... | 5.25 | |
| 46 | .... | .... | 5.55 | 5.00 | 5.55 | .... | 5.80 | | | |
| 47 | .... | .... | 5.55 | 4.20 | 4.55 | 4.10 | 4.75 | .... | 5.85 | |
| 48 | .... | .... | 5.55 | 4.75 | 5.00 | .... | 5.80 | .... | 5.65 | |
| 49 | .... | .... | 8.15 | 6.04 | 6.00 | 6.25 | 6.65 | | | |
| 50 | .... | .... | 5.25 | 4.35 | 5.15 | 5.00 | 5.85 | .... | .... | 5.65 |
| 51 | .... | .... | 5.00 | 4.75 | 4.80 | 5.65 | 5.35 | .... | .... | 5.00 |
| 52 | .... | .... | 5.55 | 5.00 | 5.00 | 5.25 | 6.00 | | | |
| 53 | .... | .... | 4.80 | 3.80 | 4.25 | 5.35 | 5.55 | | | |
| 54 | .... | .... | 6.15 | 5.25 | 5.00 | 5.00 | 5.65 | .... | .... | 5.85 |
| 55 | .... | .... | 4.75 | 3.02 | 4.10 | 4.75 | 4.70 | .... | .... | 4.15 |
| 56 | .... | .... | 6.04 | 5.85 | 6.15 | 7.10 | 6.25 | .... | .... | 6.25 |
| 57 | .... | .... | 5.55 | 5.00 | 4.70 | 5.25 | 5.85 | .... | .... | 5.86 |
| 58 | .... | .... | 4.75 | 4.35 | 4.75 | 5.00 | .... | .... | .... | 5.25 |
| 59 | .... | .... | 6.15 | 6.65 | 5.85 | 5.80 | 6.25 | .... | 6.15 | 5.75 |
| 60 | .... | .... | 4.00 | 3.80 | 5.25 | 5.35 | 5.00 | 5.55 | | |
| 61 | .... | .... | 5.75 | 5.00 | 5.00 | 5.15 | 6.15 | .... | .... | 5.80 |
| 62 | .... | .... | 5.85 | 5.55 | 5.65 | 7.10 | 5.85 | .... | .... | 5.55 |
| 63 | .... | .... | 2.30 | 3.33 | 4.80 | 4.10 | 5.25 | .... | .... | 5.85 |
| 64 | .... | .... | 4.80 | 5.00 | 5.85 | 5.00 | 5.75 | .... | .... | 6.15 |
| 65 | .... | .... | 5.55 | 4.75 | 5.00 | 5.85 | 5.00 | | | |
| 66 | .... | .... | 6.65 | 5.25 | 5.85 | 6.65 | 7.10 | | | |
| 67 | .... | .... | 5.35 | 5.00 | 5.25 | 4.75 | 5.00 | .... | .... | 5.85 |
| 68 | .... | .... | 7.10 | 6.15 | 6.65 | 5.35 | .... | .... | .... | 6.65 |
| 69 | .... | .... | 5.25 | .... | 4.75 | 5.00 | 5.25 | 5.85 | .... | 5.00 |
| 70 | .... | .... | .... | 5.55 | 5.85 | 5.00 | 5.55 | | | |
| 71 | .... | .... | .... | 5.25 | 5.00 | 5.00 | 5.85 | | | |
| 72 | .... | .... | .... | 4.75 | 5.00 | 5.25 | 5.00 | .... | .... | 5.85 |
| 73 | .... | .... | .... | 5.00 | 5.35 | 5.85 | Died | | | |
| 74 | .... | .... | .... | 5.25 | 5.85 | 6.15 | 5.85 | | | |
| 75 | .... | .... | .... | 3.80 | 4.70 | 5.00 | 5.00 | 5.35 | | |
| 76 | .... | .... | .... | 5.55 | 5.85 | 5.75 | 5.00 | 5.25 | | |
| 77 | .... | .... | .... | 4.00 | 4.00 | 5.25 | Died | | | |
| 78 | .... | .... | .... | 4.75 | 5.25 | 5.00 | 5.55 | | | |

* In the table the readings are in milligrams per hundred cubic centimeters of serum.

TABLE 1.—*Monthly Determinations of the Inorganic Phosphorus in the Blood of Children *—Continued*

| | 1925 and 1926 | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Case | Nov. | Dec. | Jan. | Feb. | March | April | May | June | Sept. | Oct. |
| 79 | .... | .... | .... | 5.35 | 5.85 | 5.25 | 5.75 | .... | 5.55 | |
| 80 | .... | .... | .... | 5.00 | 5.25 | 5.85 | 5.75 | .... | Died | |
| 81 | .... | .... | .... | 4.00 | 5.25 | 4.75 | 5.00 | 5.35 | 5.85 | |
| 82 | .... | .... | .... | 5.25 | 5.00 | 5.55 | 5.85 | | | |
| 83 | .... | .... | .... | 4.15 | 5.00 | 5.25 | 5.00 | .... | 5.35 | |
| 84 | .... | .... | .... | 5.00 | 5.35 | 5.85 | 6.15 | | | |
| 85 | .... | .... | .... | 4.10 | 5.00 | 5.75 | 5.00 | | | |
| 86 | .... | .... | .... | 3.70 | 4.55 | 4.75 | 5.00 | | | |
| 87 | .... | .... | .... | 4.10 | 5.00 | 5.25 | 5.00 | .... | 5.55 | |
| 88 | .... | .... | .... | 4.75 | 5.25 | 5.55 | 5.55 | | | |
| 89 | .... | .... | .... | 5.00 | 4.75 | .... | 5.25 | 5.00 | 5.35 | |
| 90 | .... | .... | .... | 5.25 | 5.55 | 5.55 | 6.25 | | | |
| 91 | .... | .... | .... | 4.75 | 5.55 | 6.25 | 6.65 | 7.10 | | |
| 92 | .... | .... | .... | 4.00 | 5.25 | 5.55 | .... | .... | 6.15 | |
| 93 | .... | .... | .... | 3.25 | 4.55 | 4.75 | 5.55 | | | |
| 94 | .... | .... | .... | 4.55 | 4.75 | 5.85 | 5.55 | 6.15 | | |
| 95 | .... | .... | .... | 4.30 | 4.75 | 5.25 | 5.55 | | | |
| 96 | .... | .... | .... | 4.55 | 5.00 | .... | 5.25 | | | |
| 97 | .... | .... | .... | 5.35 | 5.85 | 5.85 | 5.25 | .... | 5.00 | |
| 98 | .... | .... | .... | 5.85 | 5.25 | 5.55 | 5.85 | 5.75 | | |
| 99 | .... | .... | .... | 4.15 | 5.25 | 5.35 | 5.00 | | | |
| 100 | .... | .... | .... | 5.00 | 4.75 | 5.25 | 5.85 | | | |
| Average | 5.68 | 5.34 | 5.29 | 4.77 | 5.19 | 5.38 | 5.62 | 5.69 | 5.73 | 5.71 |

* In the table the readings are in milligrams per hundred cubic centimeters of serum.

true for both summer and winter. There was also noted a strong tendency toward citrous fruits and meat in the diets of these mothers. Forty-seven of the fifty-three nursing mothers rendered similar reports in this respect. The diets of the twenty-six older children were not quite so good in the matter of green vegetables. One of the three cases of active rickets among the breast-fed infants occurred in one whose mother ate large quantities of green vegetables, but in the other two cases the mothers had poor appetites and ate sparingly. One case of severe rickets (a selected case), presenting marked deformity of the bones of both legs, was represented in a negro boy, aged 3 years, who did not eat green vegetables and had always subsisted on a condensed milk ration. The home life and environment of this child were miserable, and he had had the poorest of general care.

Analysis of the living conditions showed that 14 per cent of the residences were exposed to the sun on all four sides; 31 per cent on three sides; 42 per cent on two sides; 11 per cent on one side, and 2 per cent were not exposed to the sun. The reports showed that 96 per cent of the children had either front or back yards for playgrounds, and many of them had both. Four per cent of the group played in alley ways or in the streets. The average number represented in each family was approximately six. Fifty-four per cent of the children had fairly comfortable sleeping quarters, while the remaining 46 per cent fared badly in this respect.

There was a direct relationship between the content of inorganic phosphorus in the blood and the percentage of possible sunshine for

TABLE 2.—*Calcium and Phosphorus Products, Determined at the Time of Admission to the Clinic*

| Case | Initial Phosphorus Determinations | Initial Calcium Determinations | Calcium and Phosphorus Products | Case | Initial Phosphorus Determinations | Initial Calcium Determinations | Calcium and Phosphorus Products |
|---|---|---|---|---|---|---|---|
| 1 | 6.65 | 11.10 | 73.81 | 51 | 5.00 | 10.30 | 51.50 |
| 2 | 6.25 | 9.09 | 56.81 | 52 | 5.55 | 11.00 | 61.05 |
| 3 | 3.65 | 10.00 | 36.50 | 53 | 4.80 | 9.90 | 47.52 |
| 4 | 7.65 | 8.33 | 63.72 | 54 | 6.15 | 8.70 | 53.50 |
| 5 | 5.90 | 9.52 | 56.17 | 55 | 4.75 | 10.50 | 49.87 |
| 6 | 4.65 | 10.52 | 47.86 | 56 | 6.04 | 10.30 | 62.21 |
| 7 | 5.90 | 12.50 | 73.75 | 57 | 5.55 | 11.20 | 63.16 |
| 8 | 7.10 | 11.80 | 83.78 | 58 | 4.75 | 10.50 | 49.87 |
| 9 | 6.25 | 9.52 | 59.50 | 59 | 6.15 | 10.80 | 66.42 |
| 10 | 6.65 | 9.09 | 60.49 | 60 | 4.00 | 10.60 | 42.40 |
| 11 | 8.30 | 10.63 | 88.22 | 61 | 5.75 | 9.60 | 55.20 |
| 12 | 8.30 | 8.73 | 72.46 | 62 | 5.85 | 11.20 | 65.52 |
| 13 | 3.03 | 8.33 | 25.24 | 63 | 2.30 | 10.50 | 24.15 |
| 14 | 4.00 | 8.00 | 32.00 | 64 | 4.80 | 8.90 | 48.72 |
| 15 | 2.30 | 7.43 | 17.09 | 65 | 5.55 | 10.70 | 59.38 |
| 16 | 5.00 | 10.40 | 52.20 | 66 | 6.65 | 11.60 | 77.14 |
| 17 | 7.65 | 10.60 | 81.09 | 67 | 5.35 | 10.20 | 64.57 |
| 18 | 7,10 | 11.00 | 78.10 | 68 | 7.10 | 10.30 | 73.13 |
| 19 | 7.10 | 10.80 | 76.68 | 69 | 5.25 | 10.40 | 54.60 |
| 20 | 5.00 | 11.00 | 55.00 | 70 | 5.55 | 11.00 | 55.55 |
| 21 | 4.50 | 11.20 | 50.40 | 71 | 5.25 | 9.60 | 50.40 |
| 22 | 6.65 | 10.20 | 67.83 | 72 | 4.75 | 10.00 | 47.50 |
| 23 | 7.10 | 8.40 | 59.64 | 73 | 5.00 | 11.00 | 55.00 |
| 24 | 6.65 | 9.80 | 65.17 | 74 | 5.25 | 8.90 | 46.72 |
| 25 | 6.65 | 7.80 | 51.87 | 75 | 3.80 | 10.20 | 38.76 |
| 26 | 5.00 | 11.60 | 58.00 | 76 | 5.55 | 10.30 | 57.16 |
| 27 | 5.25 | 10.40 | 54.60 | 77 | 4.00 | 11.00 | 44.00 |
| 28 | 7.10 | 8.00 | 56.80 | 78 | 4.75 | 9.00 | 42.75 |
| 29 | 4.55 | 11.00 | 50.05 | 79 | 5.35 | 7.40 | 39.59 |
| 30 | 4.00 | 11.60 | 46.40 | 80 | 5.00 | 7.40 | 37.00 |
| 31 | 5.55 | 10.40 | 57.72 | 81 | 4.00 | 10.60 | 42.40 |
| 32 | 6.65 | 11.20 | 74.48 | 82 | 5.25 | 11.40 | 69.86 |
| 33 | 8.15 | 8.40 | 68.46 | 83 | 4.15 | 8.40 | 34.86 |
| 34 | 6.25 | 9.40 | 58.75 | 84 | 5.00 | 9.20 | 46.00 |
| 35 | 4.55 | 10.10 | 45.95 | 85 | 4.10 | 10.00 | 41.00 |
| 36 | 5.00 | 9.30 | 46.50 | 86 | 3.70 | 11.50 | 41.55 |
| 37 | 5.25 | 11.50 | 60.37 | 87 | 4.10 | 8.60 | 35.26 |
| 38 | 4.30 | 10.90 | 46.87 | 88 | 4.75 | 11.00 | 52.25 |
| 39 | 4.15 | 8.70 | 36.10 | 89 | 5.00 | 10.20 | 51.00 |
| 40 | 4.55 | 8.00 | 36.40 | 90 | 5.25 | 10.00 | 62.50 |
| 41 | 5.55 | 12.00 | 66.60 | 91 | 4.75 | 9.60 | 45.60 |
| 42 | 5.55 | 9.30 | 51.84 | 92 | 4.00 | 10.60 | 42.40 |
| 43 | 4.35 | 10.50 | 45.66 | 93 | 5.25 | 11.50 | 60.37 |
| 44 | 4.65 | 10.30 | 47.89 | 94 | 4.55 | 10.00 | 45.50 |
| 45 | 4.35 | 8.10 | 35.23 | 95 | 4.30 | 11.20 | 48.16 |
| 46 | 5.55 | 9.70 | 53.83 | 96 | 4.55 | 9.80 | 44.59 |
| 47 | 5.55 | 10.60 | 58.83 | 97 | 4.35 | 8.60 | 37.41 |
| 48 | 5.55 | 11.50 | 63.82 | 98 | 5.85 | 10.40 | 80.84 |
| 49 | 8.15 | 10.00 | 80.15 | 99 | 4.15 | 9.60 | 39.84 |
| 50 | 5.25 | 11.30 | 59.32 | 100 | 5.00 | 9.00 | 45.00 |

TABLE 3.—*Comparative Climatologic Data Between New Orleans, New York, Denver and Los Angeles, from December, 1925, to November, 1926*

| | New Orleans | | | New York | | | Denver | | | Los Angeles | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Month | Possible Sunshine, % | Mean Temperature | Precipitation in Inches | Possible Sunshine, % | Mean Temperature | Precipitation in Inches | Possible Sunshine, % | Mean Temperature | Precipitation in Inches | Possible Sunshine, % | Mean Temperature | Precipitation in Inches |
| December, 1925.... | 50 | 52.3 | 3.58 | 58 | 34.5 | 3.33 | 55 | 32.1 | 0.93 | 77 | 63.2 | 2.45 |
| January, 1926..... | 46 | 52 | 6.10 | 57 | 31.8 | 2.52 | 56 | 31.2 | 0.71 | 87 | 59.4 | 3.06 |
| February, 1926.... | 70 | 58.8 | 3.02 | 59 | 29.4 | 5.46 | 67 | 39 | 0.40 | 75 | 61.2 | 2.70 |
| March, 1926..... | 49 | 57.6 | 20 | 79 | 35.1 | 2.59 | 60 | 37.6 | 1.98 | 57 | 63 | 0.22 |
| April, 1926..... | 67 | 67 | 6.39 | 70 | 46 | 1.78 | 61 | 47.6 | 2.31 | 42 | 63.4 | 7.53 |
| May, 1926..... | 75 | 74.8 | 13.7 | 80 | 58.5 | 2.45 | 61 | 58.6 | 1.41 | 67 | 65.4 | 0.18 |
| June, 1926..... | 72 | 81.8 | 3.70 | 62 | 64.8 | 2.47 | 65 | 66.6 | 0.86 | 55 | 67.2 | 0 |
| July, 1926..... | 69 | 83.1 | 4.20 | 67 | 73.2 | 7.47 | 57 | 70.8 | 1.00 | 72 | 69.8 | 0 |
| August, 1926..... | 64 | 83.6 | 7.25 | 58 | 72.8 | 7.57 | 63 | 73 | 0.80 | 78 | 72.6 | 0 |
| September, 1926... | 72 | 82.6 | 6.01 | 71 | 64.8 | 5.51 | 65 | 61.6 | 1.48 | 82 | 68 | 0 |
| October, 1926..... | 69 | 74 | 4.48 | 56 | 54.2 | 5.11 | 75 | 53.6 | 0.69 | 81 | 67.6 | 0.27 |

each month (chart 1). A rise in the percentage of possible sunshine was followed within about one month by a rise in the inorganic phosphorus level of the blood. A similar trend was also noted between the phosphorus curve and the temperature curve. But this is considered incidental, since the temperature curve would necessarily be governed more or less by the amount and intensity of the sunshine (chart 2). There is no relationship between the phosphorus curve and the curve which represents the monthly amount of rainfall in inches (chart 3).

In table 3, which is a comparison between the amount of possible sunshine, the mean temperature and the amount of precipitation for the cities of New Orleans, New York, Denver and Los Angeles, it will be noted that New Orleans and New York had about the same percentage of possible sunshine. The incidence of rickets in New Orleans among children aged 3 years or under is about 45 per cent,[31] whereas in New York, it is variously stated by different authors to be from 50 to 75 per cent. The severity of the disease is much more marked in New York than in New Orleans. Denver, a city of much higher altitude than either New Orleans or New York, having about the same latitude as New York, has a lower percentage of possible sunshine than either of these two cities, but with a decidedly lower incidence of rickets (26 per cent), according to Forbes and Green.[8] These authors make no reference as to the severity of the disease in Denver. Los Angeles, slightly north of New Orleans in latitude, has a considerably higher percentage of possible sunshine than either of the other three cities, with a rather constantly mild mean annual temperature. A reference to the incidence and severity of rickets in Los Angeles could not be found in the literature, but personal correspondence with Dr. Oscar Reiss of that city places the incidence at about 30 per cent, with the statement: "I do not recall ever having seen a severe case of rickets here."

## COMMENT

It is reasonably certain that the incidence of rickets would have been considerably above 26 per cent if all the children of the group had been less than 2 years of age. The incidence of the disease among the breast-fed members of the group was 5.66 per cent, while among the bottle-fed members it reached 57.1 per cent. From a consideration of the breast-fed infants, the conclusion is reached that the general health and state of nutrition of the mother, the quality of her diet and the care she gives herself and her child, are factors which largely control the presence or absence of the disease in such cases.

31. Unpublished statistics, Touro Infirmary.

A comparison of the results of the determinations of phosphorus on the blood of this group of children with the results obtained by Hess and Lundagen [20] of New York would seem to indicate that the phosphorus level in the blood of children is higher here at all times during the year than it is in New York, with a considerable difference between the winter level in New Orleans and the winter level there. These authors state that the winter level in New York is about 3.92 mg. per cent and that the summer level is about 4.34 mg. per cent. The difference between the winter and summer phosphorus levels in New Orleans was less than 1 mg. per cent. This is considered significant, because it is in keeping with the mild form of rickets encountered in this locality. It might be considered possible that the routine administration of cod liver oil and sun baths to members of this group under 1 year of age affected the phosphorus level of the entire group. But, as stated previously, the phosphorus levels of many of the older children who did not receive cod liver oil were even higher than those found in quite a few of the nonrachitic nursing infants who did. The lowest individual phosphorus averages were found in the children with active rickets, with definite changes in the bones. Some of these were as low as 2.3 mg. per cent, but the calcium levels were within normal limits. This observation corroborates the view taken by Howland and Kramer,[19] that the disturbance in the normal deposition of calcium in the bones of rachitic children is usually due to a low serum phosphorus and not necessarily to a deficiency in the calcium content.

The facts that forty-seven out of the fifty-three nursing mothers ate large quantities of green vegetables, that only three out of the fifty-three breast-fed infants developed rickets, and that two of these rachitic infants belonged to mothers who did not eat green vegetables would strongly suggest a protective influence in green vegetables against rickets. The recent work of Hess and his co-workers [32] on irradiated food substances brings up a number of conjectures in connection with rickets. It would be natural to assume that if this protective influence can be imparted to food by irradiation, the same phenomenon exists in connection with the solar rays, depending on the amount and intensity of the sunshine. Many of the common articles of food grow within wide ranges of latitude, and it would be interesting to know whether there is a difference between the antirachitic properties of green vegetables grown in the New England states, for instance, and those grown in the Gulf states.

If the incidence and severity of rickets are considered geographically, it is evident that as the equator or the Far North is approached, both frequency and severity of the disease diminish. High altitudes pro-

32. Hess, A. F., et al. (footnotes 23 and 24).

duce similar results. A given point in close proximity to the equator should receive more of the direct solar rays than one more remote, the climatic conditions, altitude, etc., being equal. This would account, in a large measure, for the rarity of the disease in the tropics. Similar circumstances would prevail at high altitudes. The rarity of the disease among the inhabitants of the frigid regions is usually explained by the protective properties in meats and oils, which make up the bulk of the diet.

In the consideration of this group of cases, one could confirm a diagnosis of rickets in only a few instances, if one accepts a calcium-phosphorus product of 30 as an absolute criterion.

Table 3 bears out in large measure the statements which have been reiterated concerning the influence of latitude and altitude on the incidence and severity of rickets.

## SUMMARY

1. Severe rickets is extremely rare in New Orleans and vicinity, but a mild form of the disease prevails to the extent of about 45 per cent among children aged 3 years or less.

2. The incidence of rickets among the 100 children studied was 26 per cent.

3. The annual phosphorus level in the blood of this group of children was considerably higher than that found by Hess and Lundagen of New York.[20] A seasonal variation in the content of inorganic phosphorus in the blood of children is verified.

4. The inorganic phosphorus content of the blood of rachitic children was always low, while the calcium levels in most of these cases were within normal limits.

5. A protective influence in green vegetables against rickets is suggested.

6. The geographic location of New Orleans is considered the factor most responsible for the occurrence of the mild form of rickets in this locality. It is possible that good exposure to the sun of the tenement houses, the low type of buildings, playground facilities, the type of clothing and the character of the diet influence it somewhat.

# STUDIES IN SCARLET FEVER

## V. SKIN BLANCHING BY COMMERCIAL ANTITOXINS *

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Since the publication of a previous communication on "Extinction Tests in Scarlet Fever," [1] many articles have appeared on the blanching effect of serum from patients convalescing from scarlet fever as compared to the blanching effect produced by the serums of animals or people who had been actively immunized by injection of (1) toxic filtrates, (2) the probable etiologic streptococcus of scarlet fever or (3) the injection of a combination of both toxic filtrate and dead bacteria.

As'troe and Constantinascu [2] injected specimens of bloods from patients with scarlet fever into rabbits; six days later, a Schultz-Charlton test of the withdrawn rabbits' serum was positive; eighteen days later, a more marked reaction occurred. Levin and Parsons,[3] Birkhaug [4] and Janovici [5] among others, also showed that the serums obtained from actively immunized animals possessed blanching qualities.

The importance of the Schultz-Charlton phenomena lies in the fact that commercial antitoxin is now being sold for diagnostic purposes, the presumable inference being that as positive or negative blanching with such antitoxins denotes the presence or absence of antibody content in the patient into whom injections are made, a diagnosis of scarlet fever may or may not be made.

To be practicable, such antitoxins injected intradermally should locally neutralize (1) any type of scarlet fever rash, (2) a high percentage of these rashes and (3) the same type of scarlet fever rash in any subsequent epidemic.

Previously,[1] we obtained a high percentage of positive blanchings with normal serum, noting, however, that not all serums "are similarly potent in producing blanching, a few normal serums not presenting blanching at all."

* Received for publication, Dec. 19, 1927.

* From the Division of Contagious Diseases, Cleveland City Hospital and the Department of Pediatrics, Western Reserve University.

1. Toomey, J. A., and Nourse, J. D.: Extinction Tests in Scarlet Fever, Am. J. Dis. Child. **27**:95 (Feb.) 1924.

2. As'troe, A., and Constantinascu, E.: Extinction Power of Sera of Rabbits Inoculated with Blood from Scarlet Fever, Compt. rend. Soc. de biol. **87**:729, 1922.

3. Levin, S. J., and Parsons, J. P.: Blanching Test in Scarlet Fever; Use of Specific Streptococcus Antiserum, Am. J. Dis. Child. **30**:232 (Aug.) 1925 .

4. Birkhaug, K. E.: Studies Concerning Blanching Phenomenon in Scarlet Fever, J. Clin. Investigation **1**:273 (Feb.) 1925.

5. Janovici, M.: The Extinction Phenomenon in Scarlet Fever, Rev. med. del Rosario, **13**:200, 1923.

We also obtained a shade better blanching with normal serum than with convalescent scarlet fever serum, but at that time we did not have the Dick test to rule out those cases which probably would have sufficient antibody content to produce blanching. This is especially true when it is recalled that the normal serum in practically all of the cases used in our previous experiments was obtained from adults, a high percentage of whom are ordinarily nonsusceptible to scarlet fever and whose serum probably would possess blanching qualities.

Commercial scarlet fever antitoxin has blanching power, but the variability of the reactions obtained led us to investigate this power further. We soon learned that antitoxin, normal human serum, etc., would not produce consistent blanching in a well defined papulated type of scarlet fever. For this reason, in all of the cases in the following experiments in which injections have been made, a rash has been present, the predominant lesion of which was a macule with barely appreciable papulation.

Objections based on differences of individual immunity can always be raised when one compares the blanching effect of one antitoxin in one person with the results obtained by injecting the same material into another person; therefore in most of the experiments, two or more antitoxins were injected into the same patient at the same time.

For the purposes of comparison, the reactions in the experiments made had the following value: negative, no blanching; plus-minus, suggestive of blanching; plus, 0.5 cm. to 1.0 cm.; 2 plus, 1.0 cm. to 1.5 cm.; 3 plus, 1.5 cm. to 2.5 cm., and 4 plus, 2.5 cm. or more of blanching. These reaction values are used for the sake of comparison only and do not take into consideration the depth of the blanching. For practical purposes, blanching would be considered present whether the reaction was 1 plus or 4 plus, 0.5 cm. or 5.0 cm. All injections were made intradermally in patients with acute scarlet fever whose rash was not over twenty-four hours old, the amount injected being 0.5 cc., unless otherwise noted.

## RESULTS

The blanching, which follows locally injected antitoxins in about one half of the cases, does not always persist as long as the rash. Antitoxins, comparatively, have varying blanching qualities; some cause local hives after the injection. The original material obtained from Drs. George and Gladys Dick and from Dr. A. R. Dochez showed definite and consistent blanching (table 1), and the latter serums gave better blanching than convalescing serums obtained from fifteen to twenty days after the onset of scarlet fever (table 2).

The treatment of a patient with convalescent serum did not disturb the efficacy of intradermal tests (table 3).

In many of the patients treated with antitoxin, there was local redness around the sites of subsequent intradermal injections of antitoxins, but good blanching occurred in others (table 4).

It might be stated that previous administration of therapeutic antitoxin would have some effect on the results of subsequent intradermal injections, but if tables 5 and 6 are studied, it is seen that local redness may occur in patients when previous antitoxin therapy had not been given.

TABLE 1.—*Reactions to Antitoxin* *

| Patient | Antitoxin | 24 Hours | 48 Hours | 72 Hours | 96 Hours | Rash |
|---|---|---|---|---|---|---|
| B. R. | Dochez | ++ | ++ | ++ | ++ | Still present |
| | Dick | +++ | +++ | +++ | +++ | Still present |
| B. W. | Dick | ++++ | ++++ | ++++ | ++++ | Still present |
| | Dochez | ++++ | ++++ | Red center | Fading | Still present |
| | Parke Davis | +++ | +++ | ++++ | Hives ++++ | Still present |
| | | | | — | — | |
| I. T. | | Papulated rash | | | | |
| | Parke Davis | ± | ± | — | — | Still present |
| | Dick | ++++ | +++ | ++ | + | Still present |
| | Dochez | ± | + | ± | — | Still present |
| I. R. | | Papulated rash | | | | |
| | Parke Davis | ± | ± | — | — | Still present |
| | Dick | + | ++ | ± | ± | Still present |
| | Dochez | + | + | ± | ± | Still present |
| J. R. | | Papulated rash | Halo | | | |
| | Parke Davis I | + | + | — | — | Still present |
| | Parke Davis II | ± | ± | — | — | Still present |
| | Horse serum, Mulford | ± | — | — | — | Still present |
| Dr. H. | | | With hives | With hives | | |
| | Dochez | + | ++++ | ++++ | Hives | Still present |
| | Dick | + | + | — | — | Still present |
| | Parke Davis | — | — | — | ++++ | Still present |
| B. C. | Dochez | — | — | + | + | Still present |
| | Dick | — | — | ++++ | ++++ | Still present |
| | Parke Davis | — | Redness, 1.5 cm. | Redness, 5 cm. | Redness, 5 cm. | Still present |
| J. Cur. | Dochez | — | ++++ | ++++ | +++ | Still present |
| | Dick | — | ++++ | ++++ | ++++ | Still present |
| | Parke Davis | — | ± | ± | Redness, 7.5 cm. | Still present |
| Carl S. | | Papulated rash | | | | |
| | Dochez | — | + | — | — | Still present |
| | Dick | — | + | — | — | Still present |
| | Parke Davis | — | + | — | — | Still present |

* No rash was over twenty-four hours old. The injected amount in each instance was 0.5 cc.

When one patient was injected with several antitoxins at the same time, treatment having not been previously given, the reactions were inconsistent, even though by dilution an effort was made to inject approximately the same number of antitoxin skin test doses into each patient (table 6).

Some antitoxins did not cause blanching the first day, but showed blanching later on the second or third day; blanching did not occur at all in some patients. Occasionally, good blanching may be obtained by subcutaneous injections (table 7).

Some old unconcentrated commercial antitoxins gave better reactions than the concentrated antitoxins of the same firm.

TABLE 2.—*Reactions to Convalescent Serums*

| | | 24 Hours | 48 Hours |
|---|---|---|---|
| **Patient B. McS.** | | | |
| (Serums from | 1. | — | +++ |
| patients with | 2. | ++ | — |
| scarlet fever, | 3. | — | ± |
| 1 to 7 inclu- | 4. | — | + |
| sive) | 5. | — | — |
| | 6. | — | — |
| | 7. | — | — |
| Control | | | |
| Dochez | | +++ | ++++ |
| Dick | | +++ | +++ |
| **Patient C. H.** | | | |
| (Serums from | 8. | ++ | +++ |
| patients with | 9. | +++ | ++ |
| scarlet fever, | 10. | ++ | ++ |
| 8 to 10 inclusive) | | | |
| Control | | | |
| Dochez | | ++++ | ++++ |
| Dick | | ++++ | ++++ |
| **Patient R. C.** | | | |
| (Serums from | 11. | ++ | — |
| patients with | 12. | ++ | + |
| scarlet fever, | 13. | ++ | ++ |
| 11 to 13 inclusive) | | | |
| Control | | | |
| Dochez | | ++ | ++ |
| Dick | | +++ | ++++ |
| **Patient M. B.** | | | |
| (Serums from | 14. | + | ++ |
| patients with | 15. | ++ | ++ |
| scarlet fever, | 16. | ++ | +++ |
| 14 to 17 inclusive) | 17. | + | + |
| Control | | | |
| Dochez | | ++++ | ++++ |
| Dick | | ++++ | ++++ |
| **Patient K. N.** | | | |
| (Serums from | 18. | +++ | +++ |
| patients with | 19. | ± | ± |
| scarlet fever, | 20. | ± | ± |
| 18 to 21 inclusive) | 21. | + | — |
| Control | | | |
| Dochez | | ++++ | ++++ |
| Dick | | ++++ | ++++ |
| **Patient E. H.** | | | |
| (Serums from | 22. | ± | ± |
| patients with | 23. | — | — |
| scarlet fever, | 24. | +++ | ++ |
| 22 to 26 inclu- | 25. | — | — |
| sive) | 26. | ++ | — |
| Control | | | |
| Dochez | | + | ++++ |
| Dick | | ++++ | ++++ |
| **Patient W. M.** | | | |
| (Serums from | 27. | — | ± |
| patients with | 28. | — | + |
| scarlet fever, | 29. | +++ | +++ |
| 27 to 33 inclu- | 30. | — | ++ |
| sive) | 31. | +++ | +++ |
| | 32. | ++ | ++ |
| | 33. | ++ | ++ |
| Control | | | |
| Dochez | | +++ | +++ |
| Dick | | ++++ | ++++ |

| | |
|---|---|
| 1. Number of cases injected | 7 |
| 2. Number of serums tested | 33 |
| 3. Amount injected in each instance was 1 cc. | |
| 4. Results at 48 hours | |
| Negative reaction | 9 or 27% |
| One plus reaction | 4 or 12% |
| Two plus reaction | 9 or 27% |
| Three plus reaction | 6 or 18% |
| Four plus reaction | 0 |
| Plus-minus reaction | 5 or 15% |
| Controls: Dochez or Dick positive in all instances | |

TABLE 3.—*Reaction of the Skin in Patients Treated with Convalescent Serum: Tests Being Performed Immediately After the Therapeutic Injection* *

| Patient | Serum of | 24 Hours | 48 Hours | Serum of | 24 Hours | 48 Hours |
|---|---|---|---|---|---|---|
| H. S. | V. O. | + | + | M. H. | + | + |
| B. G. | J. K. | + | ++ | J. R. | + | + |
| R. R. | M. G. | ++ | ++ | M. O. | + | + |
| M. L. | A. P. note 1 | ++++ | ++++ | V. O. | + | ++ |
| G. L. | A. P. note 1 | + | ++ | M. F. | + | ++ |
| S. M. | A. P. note 1 | ++ | ++ | X. | + | ++ |
| R. C. | H. B. | + | + | J. K. | + | ++ |

* All injections were in 0.5 cc. amounts. The test material was convalescent serum. NOTE 1. The serum in this instance was hemolyzed and had been inactivated for three hours so that it was extremely thick and almost gelatinous on injection.

TABLE 4.—*Skin Reactions in Patients Treated with Antitoxins, Tests Being Performed Immediately After the Therapeutic Injection* *

| | | Skin Tested with | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|
| | | Antitoxin, Dick | | Antitoxin, Dochez | | Antitoxin, Mulford | | Antitoxin, Convalescent Serum | |
| Patient | Therapeutic Antitoxin | 24 Hrs. | 48 Hrs. | 24 Hrs. | 48 Hrs. | 24 Hrs. | 48 Hrs. | 24 Hrs. | 48 Hrs. |
| F. B. | Convalescent serum, intravenously | Red papule | + | ++ | ++ | — | — | ++ | ++ |
| S. D. | Convalescent serum, intravenously | Red popule | Redness 2.5 cm. | + | Red papule | — | — | ++ | + |
| H. V. | Dick's antitoxin, intravenously | Redness | Redness, halo of blanching, 2 cm. | Wheal | Redness, halo of blanching, 1 cm. | — | — | — | — |
| S. R. | Dick's antitoxin, intramuscularly | Redness 2 cm. | Redness 2 cm. | Redness 1 cm. | Redness 1 cm. | — | — | — | — |
| M. W. | Lilly's antitoxin, intramuscularly | ± | — | — | — | — | — | — | — |
| J. B. | Dick's antitoxin, intravenously | Red indurated | +++ | +++ | ++ | ± | + | +++ | ++ |
| F. J. | Dick's antitoxin, intravenously | ± | — | — | — | — | — | — | — |
| M. P. | Dick's antitoxin, intramuscularly | +++ | ++++ | Hives | ++++ | Hives | ± | ++++ | ++++ |
| G. R. | Dick's antitoxin, intravenously | Redness 1 cm., halo of blanching | Redness 1 cm. | — | — | — | — | — | — |
| J. K. | Dick's antitoxin, intravenously | + | + | ± | — | — | — | ± | — |
| E. Y. | Dick's antitoxin, intravenously | Redness 1 cm., blanching ++ | Same | + | + | — | — | + | + |
| M. B. | Lilly's antitoxin, intramuscularly | ± | Redness 1 cm. | ± | Redness 1 cm. | — | — | ± | ± |
| A. M. | Dick's antitoxin, intravenously | Red 1 cm., blanching ++ | Redness 2 cm. | Redness 1 cm., blanching + | — | Red ±, blanching + | Redness ± | — | — |
| H. S. | Dick's antitoxin, intramuscularly | ± | — | ± | — | — | — | ± | — |
| T. V. | Dick's antitoxin, intravenously | Red 3 cm. | Red 1 cm. | Red 2 cm. | Red 1 cm. | — | — | Red 1 cm. blanching + | — |
| M. D. | Dick's antitoxin, intravenously | ± | Red 1 cm. | ± | Red 0.5 cm. | — | — | — | — |
| A. M. | Lilly's antitoxin, intramuscularly | Redness 2 cm. | Redness 2 cm. | Redness 1 cm. | Redness | 1 cm. | — | — | — |
| L. D. | Lilly's antitoxin, intramuscularly | Redness 2 cm. | Redness 2 cm. | Redness 1 cm. | — | Redness 1 cm | — | — | — |
| A. T. | Dick's antitoxin, intravenously | Redness 1 cm. | Redness 1 cm. | ± | — | ± | — | — | — |
| M. W. | Lilly's antitoxin, intramuscularly | +++ | +++ | ++++ | ++++ | — | — | — | — |
| | Parke Davis | ++ | ++ | — | — | — | — | — | — |

* All injections were in 1 cc. amounts.

The Dick antitoxins used in this experiment were received from Drs. George and Gladys Dick. The Dochez antitoxin was sent to us at our request, from Dr. A. R. Dochez.

TABLE 5.—*Reactions to Several Antitoxins Injected into the Same Patient to Whom Treatment Had Not Been Given*

| Patient | Antitoxin Injected | 24 Hrs. | 48 Hrs. |
|---|---|---|---|
| R. F. | 1. Mulford's | — | — |
| | 2. Lilly's unconcentrated | — | — |
| | 3. Dick's | ++++ | ++++ |
| | 4. Diphtheria globulin | — | — |
| | 5. Lederle's | — | — |
| | 6. Squibb's | — | — |
| | 7. Lilly's concentrated | — | — |
| | 8. Parke Davis | Redness 2.5 cm. | Redness 2.5 cm. |
| E. F. | 1. Squibb's | ++++ | ++++ |
| | 2. Lederle's | + | + |
| | 3. Mulford's | + | + |
| | 4. Lilly's concentrated | ++ | ++ |
| | 5. Parke Davis | ++ | ++ |
| | 6. Dick's | ++ | ++ |
| | Ten days later local urticaria at the site of injections 1, 2, 3; generalized urticaria on the eleventh day | | |
| J. S. | 1. Squibb's | +++ | ++ central redness |
| | 2. Mulford's | +++ | — |
| | 3. Lilly's unconcentrated | ++ | — |
| | 4. Dick's concentrated | ++++ | ++++ red center |
| | 5. Diphtheria globulin | — | — |
| | 6. Parke Davis | — | — |
| | 7. Lilly's concentrated | — | — |
| | 8. Lederle's | — | Redness + |

| Patient | Antitoxin Injected | 24 Hrs. | 48 Hrs. |
|---|---|---|---|
| A. B. | 1. Dick's | — | — |
| | 2. Mulford's | — | — |
| | 3. Squibb's | — | — |
| | Note 1: Urticaria about injection 6 days later | | |
| M. J. | 1. Dick's | Redness 0.5 cm. | Redness 0.5 cm. +++ blanching |
| | 2. Lederle's | Redness | + |
| | 3. Lilly's unconcentrated | ++++ | ++++ |
| | 4. Lilly's concentrated | Redness 0.5 cm. | ± |
| | 5. Mulford's | Redness 0.5 cm. | ++++ |
| | 6. Parke Davis | Redness 2.5 cm. | Redness 2.5 cm. |
| | 7. Squibb's | Redness 0.5 cm. | ++ |
| A. A. | 1. Dick's | ++++ 6 cm. | ++++ 6 cm. |
| | 2. Lilly's concentrated | ++++ 5.5 cm. | ++++ 5.5 cm. |
| | 3. Lilly's unconcentrated | ++++ 7 cm. | ++++ 7 cm. |
| | Note: Blanching in each case disappeared two days before rash | | |
| E. S. | 1. Dick's concentrated | — | Redness ++ |
| | 2. Parke Davis | — | Redness ++++ |
| | 3. Mulford's | — | Redness ++++ |
| | 4. Lilly's concentrated | — | — |
| | 5. Squibb's | — | — |
| | Local urticaria 17 days afterward | | |

* All amounts injected 0.5 cc.

TABLE 6.—*Summary of Reactions to the Injection of Several Antitoxins into the Same Patient and into Patients Tested (Additional to Table 5)* *

| | Blanching | | | | | | Redness—Inflammation | | | | | | Hives | Number Times |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | — | ± | + | ++ | +++ | ++++ | — | ± | + | ++ | +++ | ++++ | Local | Tested |
| Mulford's | 11 | 0 | 3 | 0 | 0 | 1 | 14 | 0 | 1 | 0 | 0 | 0 | 3 | 15 |
| Lilly's unconcentrated | 2 | 0 | 1 | 2 | 1 | 8 | 14 | 0 | 0 | 0 | 0 | 0 | 4 | 14 |
| Lilly's concentrated | 7 | 1 | 0 | 2 | 1 | 4 | 8 | 1 | 0 | 2 | 1 | 3 | 3 | 15 |
| Dick's original † | 1 | 1 | 0 | 1 | 1 | 12 | 15 | 0 | 1 | 0 | 0 | 0 | 0 | 16 |
| Lederle's | 9 | 1 | 3 | 0 | 0 | 0 | 11 | 1 | 1 | 0 | 0 | 0 | 1 | 13 |
| Squibb's | 8 | 2 | 1 | 1 | 1 | 2 | 7 | 0 | 1 | 1 | 5 | 1 | 2 | 15 |
| Parke Davis | 10 | 2 | 1 | 1 | 0 | 0 | 4 | 0 | 1 | 1 | 2 | 6 | 5 | 14 |
| Total | 48 | 7 | 9 | 7 | 4 | 27 | 73 | 2 | 5 | 4 | 8 | 10 | 18 | 102 |

| | Blanching | | Redness | |
|---|---|---|---|---|
| | Number | Per Cent | Number | Per Cent |
| Negative | 48 | 47 | 73 | 71 |
| Plus-minus | 7 | 7 | 2 | 2 |
| One plus | 9 | 9 | 5 | 5 |
| Two plus | 7 | 7 | 4 | 4 |
| Three plus | 4 | 4 | 8 | 8 |
| Four plus | 27 | 26 | 10 | 10 |

* The rash was never over twenty-four hours old and the injection in amounts of 0.5 cc. intradermally.

† Many patients tested later had local hives following the intradermal injection. This might be due to some change associated with ageing material.

TABLE 7.—*Reactions to the Same Serums When the Injections Were Made Intradermally and Subcutaneously* *

| Patient | Antitoxin | Intradermal | | Subcutaneous | | Comment |
|---|---|---|---|---|---|---|
| | | Blanching | Redness | Blanching | Redness | |
| 1 | U. S. P. | — | — | — | — | |
| 2 | U. S. P. | ± | — | — | — | Local hives |
| 3 | U. S. P. | ± | — | ± | — | |
| 4 | U. S. P. | ++++ | — | — | — | |
| 5 | U. S. P. | ++++ | — | — | ++ | Redness first; 6 hours later blanching about site of intradermal injection |
| 6 | U. S. P. | — | ++++ | — | .. | Redness; 8 days later local urticaria |
| 7 | Dick | ++++ | — | ++++ | — | Urticaria about both sites of injection later |
| 8 | Dick | ++ | — | ++ | — | |
| 9 | Dick | .. | ++++ | .. | ++++ | Urticaria about both sites of injection later |
| 10 | Dick | — | — | — | — | |
| 11 | Dick | ++++ | — | ++++ | — | |
| 12 | U. S. P. | ++ | — | — | — | |
| 13 | Lilly, lot 1 | + | ++ | + | ++++ | |
| 14 | Lilly, lot 1 | + | + | + | ++++ | |
| 15 | Lilly, lot 1 | — | — | — | — | |
| 16 | Lilly, lot 1 | ++ | — | + | — | |
| 17 | Lilly, lot 1 | — | ++++ | — | — | |
| 18 | Lilly, lot 2 | — | — | — | — | |
| 19 | Lilly, lot 2 | — | — | + | — | |
| 20 | Lilly, lot 2 | — | — | — | — | |
| 21 | Lilly, lot 2 | — | — | — | — | |
| 22 | Lilly, lot 2 | — | — | — | — | |
| 23 | Lilly, lot 2 | — | — | — | — | |
| 24 | Lilly, lot 2 | + | ++ | + | + | |
| 25 | U. S. P. | + | — | — | — | Urticaria, local, 5 days after subcutaneous injections |
| 26 | U. S. P. | — | ++++ | — | ++ | |
| 27 | U. S. P. | — | — | — | — | |
| 28 | U. S. P. | — | ++++ | — | ++++ | Redness lasted 4 days |
| 29 | U. S. P. | — | — | — | — | |
| 30 | U. S. P. | — | + | ++ | — | Redness about intradermal injection appearing 2 days later was cleared by the fifth day when site of subcutaneous injection became red, clearing up by the eighth day |
| 31 | U. S. P. | — | — | — | — | |
| 32 | U. S. P. | + | ++ | — | ++++ | |
| 33 | Lilly, lot 3 | — | ++ | — | ++ | |
| 34 | Lilly, lot 3 | — | ++ | — | ++ | |
| 35 | Lilly, lot 3 | + | ++++ | + | ++++ | |
| 36 | Lilly, lot 3 | — | ++++ | — | ++++ | |
| 37 | Lilly, lot 3 | — | ++ | — | — | |
| 38 | Lilly, lot 3 | — | — | — | — | |
| 39 | Lilly, lot 3 | + | ++ | + | ++ | |
| 40 | Lilly, lot 3 | + | ++++ | — | ++++ | Two days later slight amount of blanching about site of intradermal injection |
| 41 | Dick's original 1 year after receiving | ++++ | — | — | — | Had received full therapeutic dose the same day |
| 42 | Same as 41 | ++++ | — | — | — | Same as 41 |
| 43 | 11, only 0.1 cc. | — | — | — | — | Urticaria 7 days later about the blanching |

* The amount injected was always 0.5 cc.

## COMMENT

If one insists that blanching is a sine qua non of a scarlet fever antitoxin, then the fact that blanching does not occur locally with many antitoxins suggests either that the antitoxin is not of specific value and achieves whatever good it accomplishes as a nonspecific foreign protein, or that it is not specific in the sense that many heterologous strains of streptococci may cause scarlet fever, against which the particular antitoxin tried is not specific. But even when antitoxins made by the use

of several strains of streptococci are tested, blanching does not result. Such observations would suggest that the mechanism of the rash is not quite clear.

The large number of patients who show local redness after intradermal injections of scarlet fever antitoxin and never any blanching is disturbing to the efficacy of the test.

Little or no attention is paid to the elapsed time between the first symptoms of the period of invasion and the eruptive period. This could, perhaps, be long enough for the production of some active immunity. It might be possible for such patients, in some degree, to become sensitive to scarlet fever antitoxin in the process of being actively immunized during an attack of the disease. As a consequence, subsequent local intradermal injection of artificial prepared antitoxin would cause local inflammation not unlike a modified Arthus phenomenon.

At least the observation of so many local red reactions and subsequent local urticaria suggests sensitivity of some kind, and, were this so, it would explain some of the severe reactions which may follow the use of scarlet fever antitoxins.

Birkhaug [4] states that epinephrine does not cause blanching of the scarlet rash, an experience not similar to ours, as we have been able to get a positive extinction test every time this drug is injected.

## CONCLUSIONS

We have tested several commercial antitoxins for their blanching qualities.

1. Many antitoxins now on the market produce little or no blanching.

2. Not all antitoxins, even if they do blanch, will blanch in the same degree when the same person is used for all the tests.

3. Even though there is blanching after injection of antitoxin, this blanching does not always last as long as the rash.

4. Many antitoxins cause local redness at the site of injection instead of blanching.

# THE RESPIRATORY METABOLISM IN INFANCY AND IN CHILDHOOD

## VIII. THE RESPIRATORY EXCHANGE IN MARASMUS: BASAL METABOLISM *

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Infantile marasmus is commonly attributed either to chronic inanition or to a derangement of tissue metabolism. According to the former and generally accepted view, the undernutrition is merely the end-result of prolonged starvation (insufficient caloric intake) or of repeated gastro-intestinal upsets (defective absorption of foodstuffs) induced by or independent of parenteral infections. A few authors,[1] however, maintain that the basic fault is a derangement of the energy metabolism resulting from defective utilization or from excessive combustion of foodstuffs in the tissues. They believe that some marantic infants fail to thrive in spite of an adequate diet and in the absence of parenteral or alimentary infection. The experimental evidence at hand is inadequate to enable one to decide finally on the merits of this view. The only systematic study of the subject was made by Fleming and Hutchinson,[2] who concluded that evidence of defective absorption or of abnormal utilization of food is not present in marasmus. Their report, however, does not include observations on the effect of food or of activity on metabolism.

The present work was undertaken to obtain evidence for or against the view that the chief cause of marasmus is an abnormal energy metabolism. The energy requirement of marantic infants was estimated in sleep, during activity (crying) and after ingestion of food. The total metabolism and the protein and carbohydrate metabolism with and without the administration of insulin were also studied. Control studies of normal infants were invariably made under comparable conditions.

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1. Marfan, A. B.: Traité de l'allaitement, Paris, 1920, p. 621. Utheim, K.: Advanced Chronic Nutritional Disturbances in Infancy, J. Metab. Research **1**:803, 1922.

2. Fleming, G. B., and Hutchison, H. S.: A Study of the Metabolism in the Undernourished Infant, Quart. J. Med. **17**:339 (July) 1924.

and differences in the metabolic response of the two groups of subjects were carefully noted. This plan of procedure consumed a great deal of time, but it seemed essential for our purpose.

This first paper considers the basal energy requirement of marantic infants. The other aspects of the respiratory exchange in marasmus will be considered in subsequent communications.

## REVIEW OF THE LITERATURE

Reference will be limited in this report to studies of the basal metabolism in marasmus. Benedict and Talbot [3] and Murlin and Hoobler [4] have reviewed the early work on the subject, and bibliographies may be found in two recent papers of Talbot.[5] This early work is generally not comparable with more modern studies because of differences in technic and inadequate control of activity during observations.

Our present knowledge of the basal metabolism in marasmus is derived mainly from the researches of Benedict and Talbot,[3] Murlin and Hoobler,[4] Fleming,[6] Fleming and Hutchinson [2] and Talbot.[7] The uniform technic and conditions of the experiments make their results mutually comparable. These observers demonstrated the presence of an accelerated basal metabolic rate per kilogram of body weight in marasmus. Fleming [8] and Talbot [7] computed the production of heat of their marantic infants on the basis of expected weight for age. They found that, on this basis, the basal rate of the infants who were 35 per cent or more underweight fell below the normal range of from 50 to 60 calories per kilogram, whereas it usually remained normal in the infants who were less than 35 per cent underweight. Tisdall and his co-workers [9] accepted the assumption of Fleming that this figure of 35 per cent represented the fraction of the total loss of weight of emaciated infants which

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3. Benedict, F. G., and Talbot, F. B.: The Gaseous Metabolism of Infants with Special Reference to Its Relation to Pulse-Rate and Muscular Activity, Carnegie Institute of Washington, Publication no. 201, 1914; Studies in the Respiratory Exchange of Infants, Am. J. Dis. Child. **8**:1 (July) 1914.

4. Murlin, J. R., and Hoobler, B. R.: The Energy Metabolism of Ten Hospital Children Between the Ages of Two Months and One Year, Am. J. Dis. Child. **9**:81 (Feb.) 1915.

5. Talbot, F. B.: Severe Infantile Malnutrition — The Energy Metabolism with a Report of a New Series of Cases, Am. J. Dis. Child. **22**:358 (Oct.) 1921; Basal Metabolism of Children, Physiol. Rev. **5**:477 (Oct.) 1925.

6. Fleming, G. B.: An Investigation Into the Metabolism in Infantile Atrophy, with Special Reference to the Respiratory Exchange, Quart. J. Med. **14**:171 (Jan.) 1921.

7. Talbot (footnote 5, first reference).

8. Fleming (footnote 2 and 6).

9. Tisdall, F. F.; Drake, T. G. H., and Brown, A.: The Carbohydrate Metabolism of the Marantic Infant, Am. J. Dis. Child. **30**:829 (Dec.) 1925.

occurred exclusively at the expense of metabolically inert tissue (fat and glycogen deposits) and calculated the average heat output of the marantic infants of Fleming,[6] Talbot[7] and Fleming and Hutchinson[2] on the basis of this additional weight. They found it to be 52.2, 58.5 and 52.5 calories per kilogram of computed weight (100/65 of the actual weight), respectively, as against an average output of heat of 54.2 calories per kilogram in well-nourished infants.

All the evidence is therefore in accord in showing an elevated basal metabolism per true weight and a diminished metabolism per expected weight in marasmus. Students of the subjects, however, are not agreed on the question as to whether the character of the fundamental metabolism is or is not changed in this condition. One group of workers believes that because of the malnutrition the active protoplasmic tissue is incapable of forming as much heat as during health; another group maintains that this tissue may produce more heat in response to an increased loss of heat, while a third group is of the opinion that the heat output per kilogram of active tissue is the same in marasmus as in health. Furthermore, the same observer frequently makes use of two or even of all three of these views to explain observed differences in the production of heat in marantic infants. Further discussion of this phase of the subject is reserved for a later section of the paper in which evidence from the literature and from the present study will be introduced in an attempt to reconcile these apparently conflicting views.

## METHODS AND SUBJECTS

The respiratory exchange was measured in an apparatus of the closed circuit type by the method of indirect calorimetry. The production of heat was calculated with the aid of the modified table of Zuntz and Schumberg[10] from the consumption of oxygen, the nonprotein respiratory quotient and the nitrogen excreted in the urine. A detailed description of the apparatus, the technic employed during the observations and the methods of calculation appeared in earlier papers of this series.[11]

10. Zuntz, N., and Schumberg: Studien zu einer Physiologie des Marches, Berlin, 1901, p. 361. Lusk, G.: Analysis of the Oxidation of Mixtures of Carbohydrate and Fat — a Correction, J. Biol. Chem. **59**:41 (Feb.) 1924.

11. Levine, S. Z., and Wilson, J. R.: The Respiratory Metabolism in Infancy and in Childhood: I. Basal Metabolism of Children, Am. J. Dis. Child. **31**:323 (March) 1926. Wilson, J. R.; Levine, S. Z., and Rivkin, H.: II. Ketosis and the Respiratory Exchange in Children, Am. J. Dis. Child. **31**:335 (March) 1926. Levine, S. Z.; Wilson, J. R.; Berliner, F., and Rivkin, H.: VI. The Specific Dynamic Action of Food in Normal Infants, Am. J. Dis. Child. **33**:722 (May) 1927. Levine, S. Z., and Wilson, J. R.: VII. The Elimination of Water Through the Skin and Respiratory Passages of Infants, Am. J. Dis. Child. **35**:54 (Jan.) 1928.

As the effect of restlessness on metabolism surpasses the trivial effect of minimal amounts of food, all the infants were given small milk meals usually containing fewer than 50 calories, from one to two hours preceding the determinations of basal metabolism (table 2). With this precaution, the infants slept soundly throughout all observations. The temperature and relative humidity ranged between 23 and 26 C. and between 35 and 60 per cent in all experiments, fluctuations in individual experiments rarely exceeding 0.5 C. and 5 per cent, respectively.

The reliability of observations was controlled by frequent alcohol checks. The detailed data appeared in papers VI and VII.[12] For 1926, the average error in the consumption of oxygen which furnishes the basis for calculating the production of heat was — 1.5 per cent, the

TABLE 1.—*Summary of Data of Subjects*

| Subject | Age, Months | Sex* | Weight, Kg. | Average Weight† For Age | Average Weight† For Height | Height, Cm. | Average Height† For Age | Surface Area,‡ Sq. M. |
|---|---|---|---|---|---|---|---|---|
| | | | | Normal Infants | | | | |
| 1 | 3 | ♂ | 6.19 | 6.06 | 7.10 | 64.5 | 61.2 | 0.341 |
| 2 | 4 | ♂ | 5.63 | 6.56 | 6.39 | 62.0 | 63.0 | 0.316 |
| 6 | 10 | ♂ | 7.56 | 9.11 | 8.80 | 71.0 | 72.4 | 0.408 |
| 9 | 1 | ♂ | 3.64 | 4.53 | 4.23 | 53.0 | 55.5 | 0.236 |
| | | | | Marantic Infants | | | | |
| 3 | 5 | ♂ | 3.49 | 7.24 | 4.77 | 55.5 | 65.4 | 0.230 |
| 4 | 5 | ♂ | 2.94 | 7.38 | 4.62 | 55.0 | 65.9 | 0.205 |
| 5 | 4½ | ♂ | 3.71 | 7.00 | 5.22 | 57.0 | 64.5 | 0.239 |
| | 6 | .. | 5.17 | 7.83 | 6.81 | 63.5 | 67.6 | 0.298 |
| | 7 | .. | 6.14 | 8.12 | 7.10 | 64.5 | 68.6 | 0.338 |
| | 7½ | .. | 6.36 | 8.28 | 7.24 | 65.0 | 69.2 | 0.350 |
| | 8 | .. | 6.79 | 8.48 | 7.44 | 65.7 | 69.9 | 0.376 |
| 7 | 3 | ♂ | 2.25 | 6.19 | .... | .... | 61.6 | 0.172 |
| 8 | 9½ | ♂ | 4.70 | 8.96 | 5.43 | 62.5 | 71.8 | 0.280 |
| 10 | 2½ | ♀ | 2.69 | 5.30 | 4.23 | 53.5 | 58.6 | 0.198 |

* In this column, ♂ indicates male; ♀, female.
† Woodbury tables, pages 106 and 107 (U. S. Dept. of Labor, Children's Bureau, pub. no. 87, 1921).
‡ Benedict and Talbot table, p. 62 (pub. no. 302, Carnegie Inst., Washington, 1921).

maximum error being — 2.8 per cent on January 8. For the following year, the average and maximum errors were — 1.6 and — 3.2 per cent, respectively. The respiratory quotients for the two years averaged 0.673 and 0.670 and ranged between 0.67 and 0.69 and between 0.66 and 0.68, as against the theoretical quotient of 0.667. It was impossible in some instances to maintain a steady alcohol flame without dissipating heat in excess of the minimal amounts produced by some of the extremely emaciated infants.

The basal metabolism of four control and six marantic infants was determined in fourteen calorimetric observations of from one to three hours' duration. They received adequate diets and were free from evident infection or alimentary disturbance throughout the course of these

12. Levine et al (footnote 11, references 3 and 4).

studies. The histories of two of the normal infants appeared in paper VI of this series (subjects 1 and 2).[13] The histories of the other infants will be presented in later papers. The nutritional condition of the subjects and the calorimetric data are summarized in tables 1 and 2.

The control infants were well nourished and approximately normal in weight and height for their ages.[14] The marantic infants were all markedly emaciated, being in every instance 47 per cent or more under-

TABLE 2.—*Calorimetric Data in Terms of Averages Per Hour*

| Subject | Date | Duration of Observation, Min. | Water, Gm. | Carbon Dioxide, Gm. | Oxygen, Gm. | Respiratory Quotient | Urinary Nitrogen, Gm. | Calories | Rectal Temperature, F. | Comment* |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | | | Normal Infants | | | | | |
| 1 | 2/ 3/26 | 80 | 6.84 | 5.21 | 4.28 | 0.89 | 0.096 | 94.5 | 98.5 | L. M. 62 cal. P. 2.6 Gm. 1 hr. p. c. |
| 2 | 4/21/26 | 90 | 6.34 | 4.67 | 3.87 | 0.88 | 0.087 | 13.1 | 98.8 | L. M. 34 cal. P. 4.0 Gm. 1 hr. p. c. |
| 6 | 11/10/26 | 163 | 8.59 | 7.33 | 5.80 | 0.92 | 0.161 | 19.8 | 98.8 | L. M. 203 cal. P. 8.1 Gm. 1¾ hr. p. c. |
| 9† | 3/14/27 | 182 | 3.46 | 2.67 | .... | .... | 0.013 | 7.77 | 98.7 | L. M. 28 cal. P. 1.3 Gm. 1 hr. p. c. |
| | | | | | Marantic Infants | | | | | |
| 3 | 2/24/26 | 121 | 4.81 | 4.45 | 3.41 | 0.95 | 0.086 | 11.7 | 97.8 | L. M. 99 cal. P. 3.3 Gm. 1 hr. p. c. |
| 4 | 4/11/26 | 60 | 4.65 | 3.64 | 2.97 | 0.89 | 0.065 | 10.1 | 97.4 | L. M. 86 cal. P. 4.0 Gm. 1 hr. p. c. |
| 5 | 11/ 1/26 | 150 | 5.57 | 4.12 | 3.37 | 0.89 | 0.078 | 11.4 | 98.6 | L. M. 43 cal. P. 2.0 Gm. 1¼ hr. p. c. |
| | 12/22/26 | 120 | 9.52 | 5.45 | 4.71 | 0.84 | 0.144 | 15.7 | 99.5 | L. M. 55 cal. P. 2.5 Gm. 1½ hr. p. c. |
| ‡ | 1/14/27 | 120 | 8.65 | 6.14 | 4.78 | 0.94 | ..... | 16.5 | 98.6 | L. M. 64 cal. P. 3.0 Gm. ¾ hr. p. c. |
| | 1/26/27 | 136 | 8.85 | 5.96 | 4.93 | 0.88 | 0.116 | 16.7 | 99.0 | L. M. 43 cal. P. 2.0 Gm. 1¼ hr. p. c. |
| | 2/ 9/27 | 75 | 10.63 | 6.29 | 5.39 | 0.85 | 0.101 | 18.2 | 98.8 | L. M. 43 cal. P. 2.0 Gm. 1 hr. p. c. |
| 7 | 12/ 6/26 | 94 | 2.83 | 2.55 | 2.13 | 0.87 | 0.022 | 7.25 | 98.0 | L. M. 43 cal. P. 2.0 Gm. 1¼ hr. p. c. |
| 8 | 12/24/26 | 120 | 8.57 | 5.63 | 4.52 | 0.91 | 0.086 | 15.4 | 98.5 | L. M. 43 cal. P. 2.0 Gm. 1 hr. p. c. |
| 10‡ | 2/14/27 | 120 | 3.05 | 2.84 | 2.37 | 0.87 | ..... | 8.06 | 98.1 | L. M. 28 cal. P. 1.3 Gm. 1 hr. p. c. |

* The subjects slept throughout all the observations. L. M. indicates last meal, p. c. indicates after feeding, P. indicates protein.

† Oxygen cylinder leaked. Heat production calculated from carbon dioxide at assumed respiratory quotient of 0.85.

‡ Calorimeter specimen lost. Assumption made that 15 per cent of total calories was derived from protein.

weight and with one exception 27 per cent or more underheight when first observed (subject 8 was only 13 per cent underheight). One infant (subject 5), who was studied at intervals over a period of three months, increased in weight from a minimum of 47 per cent to a maximum of 20 per cent below the normal weight standards for age.[14]

13. Levine et al (footnote 11, reference 3).

14. Woodbury, R. M.: Statures and Weights of Children Under Six Years of Age, U. S. Department of Labor, Children's Bureau, Publication no. 87, 1921.

## RESULTS

The results are summarized in table 3. Chart 1 shows the degree of malnutrition, expressed as percentage of expected weight for age, and the basal metabolism per kilogram of true weight and per kilogram of expected weight in accordance with the method of Talbot [7] and Fleming.[6]

The basal metabolism of three of the four control infants (subjects 1, 2 and 9) fell within the normal range of from 48 to 62 calories per kilogram per twenty-four hours. Deviations from the standard figures of Benedict and Talbot [15] were ± 0, + 1 and + 2, respectively. One

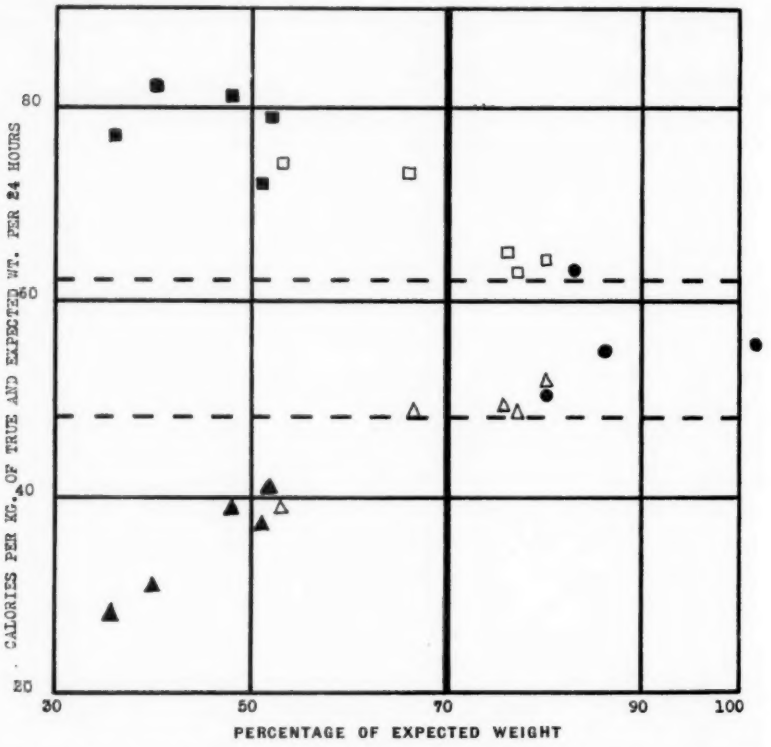


Chart 1.—The degree of malnutrition expressed as percentage of expected weight (for age), and the basal metabolism per kilogram of true weight and per kilogram of expected weight. The area within the broken lines shows the normal range of basal metabolism in infancy. The black circles represent the controls; the squares, the metabolism per actual weight of the marantic infants; the triangles, the metabolism per expected weight of the marantic infants. The white squares and triangles represent case 5.

15. Benedict, F. G., and Talbot, F. B.: Metabolism from Birth to Puberty, Carnegie Institute of Washington, Publication no. 302, 1921. Benedict, F. G.: Energy Requirements of Children from Birth to Puberty, Boston M. & S. J. **181**:107 (July 31) 1919.

infant (subject 6), whose basal rate was at the upper limit of normal (63 calories per kilogram), had received a meal considerably in excess of the maintenance requirement one and three-quarter hours preceding the observation (203 calories). The large meal undoubtedly accelerated the metabolism. On the basis of height which Talbot [16] has recently proposed as the most satisfactory single standard for normal children, the deviations from the standard figures for the four control infants were $-6$, $\pm 0$, $+2$ and $+1$ per cent, respectively. The three infants (subjects 2, 6 and 9) who were from 14 to 20 per cent underweight for age were all less than 15 per cent underweight for height, and their bodies were covered with an abundant layer of subcutaneous fat.

TABLE 3.—*Summary of Basal Metabolism Tests*

| Case | Deviation* from Average Weight for Age, per Cent | Calories, per 24 Hours | Calories, per Kg. per 24 Hours | Calories,† per Sq. M. per Hour |
|---|---|---|---|---|
| | | Normal Infants | | |
| 1 | + 2 | 349 | 56.2 | 42.6 |
| 2 | —14 | 314 | 55.8 | 41.4 |
| 6 | —17 | 475 | 62.8 | 48.5 |
| 9 | —20 | 187 | 51.2 | 32.9 |
| | | Marantic Infants | | |
| 3 | —52 | 282 | 80.7 | 51.0 |
| 4 | —60 | 242 | 82.3 | 49.2 |
| 5 | —47 | 274 | 73.9 | 47.8 |
| | —34 | 378 | 73.0 | 52.8 |
| | —24 | 396 | 64.6 | 48.9 |
| | —23 | 400 | 62.9 | 47.7 |
| | —20 | 436 | 64.2 | 48.3 |
| 7 | —64 | 174 | 77.3 | 42.2 |
| 8 | —48 | 370 | 78.8 | 55.1 |
| 10 | —49 | 194 | 71.9 | 40.7 |

* Woodbury table, page 106.
† Benedict and Talbot table, page 62.

The basal metabolic rate per kilogram of true weight was notably elevated in the group of marantic infants. Their production of heat ranged between 72 and 82 calories per kilogram per twenty-four hours and between 41 and 55 calories per square meter per hour, as against the ranges of from 48 to 62 calories per kilogram and of 33 to 43 calories per square millimeter in the normal infants (exclusive of case 6).

When calculated on the basis of expected weight for age as originally suggested by Fleming,[6] the basal rate per kilogram was invariably diminished, the extent of the reduction being directly proportional to the severity of emaciation. The most emaciated infant (subject 7) who was 74 per cent underweight for age produced a minimum of 28 calories per kilogram (of expected weight), whereas another infant (subject 8) who was 48 per cent underweight produced more than 40 calories per kilogram.

16. Talbot (footnote 5, second reference).

The results in the infant (subject 5) whose basal metabolism was estimated at intervals for three months is of special interest. When first observed he was 47 per cent below the average normal weight and at that time his production of heat per kilogram of true weight was 74 calories, but per kilogram of expected weight it was only 39.5 calories. At the end of about two months, his weight had increased so rapidly that it was less than 35 per cent below his expected weight and, although the basal rate per kilogram of true weight remained at the high level of 73 calories, it rose to the lower limit of the normal range when computed on the basis of expected weight (48 calories per kilogram). When this infant was 34 per cent underweight his basal metabolism, therefore, approximated that for a well-nourished infant of the same age. In the last three observations, when his actual weight exceeded 75 per cent of the average normal weight, the basal rate per kilogram of true as well as of expected weight attained a practically normal level (chart 1).

The respiratory quotients of the control and the marantic group of infants showed only trivial differences.

It may be briefly stated at this point that these results, which will be considered in greater detail in the following section, are in complete accord with those previously reported by Fleming [8] and Talbot.[7]

## COMMENT

All the available evidence obtained from the literature and from the present study establishes the rate of fundamental metabolism in marasmus to be as follows: In severe marasmus, the basal rate is invariably high per kilogram of true weight and invariably low per kilogram of expected weight (for age). In mild malnutrition, the basal rate on the basis of both true and expected weight is usually maintained within normal limits. The change from a normal to an augmented metabolism per true weight and to a reduced metabolism per expected weight becomes evident when the losses of weight exceed approximately 20 and 35 per cent, respectively.

Fleming [6] and other observers [17] ascribed the normal basal rate (per expected weight) of the malnourished infants who are less than 30 or 35 per cent underweight to the lowered content in the organism of fat and other metabolically inert tissue which, in their opinion, comprise approximately 35 per cent of the body of well nourished infants. The absence of this tissue from the organism probably does not modify the metabolism. This hypothesis also affords a reasonable explanation for the exaggerated output of heat per kilogram of true weight in these infants without the necessity of assuming abnormal metabolic processes.

Evidence in favor of the view that the low body weight in marasmus is due primarily to the failure of the organism to lay down fat rather

17. Footnotes 16 and 9.

than to its inability to retain nitrogen (presumably active protoplasm) is furnished by the results of Fife and Veeder [18] and Schloss.[19] Each of these authors studied the nitrogen metabolism of two emaciated infants who were on adequate diets for periods of three and two weeks, respectively, and found that the infants showed nitrogen equilibrium or positive balance during the time when their weights remained practically stationary (no deposition of fat).

Although they are not conclusive, the data on the composition of the infant's body in different states of nutrition, as compiled by various authors [20] and collected by Murlin,[4] tend to support the view that the initial failure to deposit fat and other inert tissue is the essential cause for the failure of marantic infants to thrive. In table 4, the figures for body nitrogen and for body fat, as reported by the foregoing authors, are recorded in percentages of both fresh and dried body substance. The fat content of the body served as an index of the state of nutrition, and the relative amounts of fat and nitrogenous material were compared in the normal and emaciated infants. The method of calculation on the basis of dried body substance was used in preference to the method based on fresh substance in order to eliminate the factor of body water which is notoriously subject to rapid fluctuations, especially in the infant's organism.[21]

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18. Fife, C. A., and Veeder, B. S.: Studies in the Metabolism of Atrophic Infants, Am. J. Dis. Child. **2**:19 (July) 1911.

19. Schloss, O. M.: Personal communication to the authors.

20. Camerer, W.: Analytischen Beitragen von Dr. Soldner u. Dr. Herzog. Die Chemische Zusammensetzung des neugebornen Menschen, Ztschr. f. Biol. **25**:1 (new series) 1902. Sommerfeld, P.: Zur Kenntnis der Chemischen Zusammensetzung des kindlichen Korpers im ersten Lebensjahre, Arch. f. Kinderh. **30**:253, 1900. Steinitz, F.: Ueber den Einfluss von Ernahrungsstorungen auf die Chemische Zusammensetzung des Sauglingskorpers, Jahrb. f. Kinderh. **59**:447, 1904. Steinitz, F., and Weigert, R.: Ueber den Einfluss einseitliger Ernanhrung mit Kohlehydraten auf die Chemische Zusammensetzung des Sauglingskorpers, Beitr. z. Chem. Phys. u. Path. **6**:206, 1904-1905; Ueber Demineralization und Fleischtherapie bei Tuberculose, Jahrb. f. Kinderh. **61**:147, 1905; Monatschr. f. Kinderh. **4**:301, 1905. Klose: Jahrb. f. Kinderh. **80**:154, 1914. Michel: Compt. rend. Soc. de biol. **27**:422, 1899. Fehling, Camerer: Ztschr. f. Biol. **39**:175, 1900.

21. Attention is called at this point to the results of the analyses when computed in percentages of fresh body substance. On this basis, the analyses show a fairly uniform proportion of nitrogenous material to total body weight in all of the infants, but both the normal infant of Sommerfeld and the fat infant of Steinitz and Weigert were actually considerably underweight when compared with the accepted weight standards — 30 and — 45 per cent, respectively (Woodbury, R. M.: U. S. Department of Labor, Children's Bureau Department of Labor, Pub. No. 87, 1921). It is at least conceivable that the body nitrogen expressed as percentage of fresh substance might be lower in truly normal infants.

The analyses on the basis of dried body substance show the presence of an inverse relation between body fat (inert tissue) and body nitrogen (active protoplasm). The normal infants and the one fat infant whose body fat ranged between 38 and 59 per cent of the dried substance contained an average of 6.6 per cent of nitrogen, as against an average of 11.7 per cent for the marantic infants whose body fat ranged between 7 and 20 per cent.

It is noteworthy that the relative amounts of nitrogenous material in the bodies of the emaciated infants who were all 30 per cent or more underweight for age when computed in terms of either fresh or dried

TABLE 4.—*Relation of Fat to Nitrogen Content in Body of Infants. Compiled from Various Authors*[20] *

| | | | Weight in Grams | | | In 100 Parts of Fresh Substance | | In 100 Parts of Dried Substance | |
|---|---|---|---|---|---|---|---|---|---|
| Author | Condition | Age, Months | Total | Water | Dried Substance | Fat | Nitrogen | Fat | Nitrogen |
| Steinitz and Weigert | Fat | 4 | 3,711 | 2,187 | 1,524 | 24.2 | 2.25 | 58.8 | 5.5 |
| Klose..... | Normal | Newly born | 3,210 | 2,117 | 1,093 | 17.6 | 1.91 | 51.9 | 5.6 |
| Sommerfeld | Normal | 3 | 4,340 | 3,053 | 1,287 | 13.4 | 2.27 | 44.9 | 7.6 |
| Camerer and Söldner | Normal† | Newly born | 2,821 | 2,026 | 795 | 12.3 | 1.98 | 43.8 | 7.0 |
| Michel..... | Normal | Newly born | 3,335 | 2,308 | 1,027 | 11.8 | 2.24 | 38.2 | 7.3 |
| Steinitz and Weigert | Tuberculosis and edema | 12 | 4,519 | 3,602 | 917 | 4.0 | 2.15 | 19.9 | 10.6 |
| Fehling..... | Prematurity and atrophy | Newly born | 1,496 | 1,105 | 391 | 5.1 | 2.85 | 19.5 | 10.9 |
| Steinitz and Weigert | Richets and atrophy | 12 | 2,585 | 2,102 | 483 | 2.3 | 2.3 | 12.4 | 12.3 |
| Steinitz..... | Atrophy | 2½ | 1,960 | 1,612 | 348 | 1.8 | 2.13 | 10.2 | 12.1 |
| Steinitz..... | Atrophy | 3¾ | 3,190 | 2,548 | 612 | 2.0 | 2.51 | 9.9 | 12.5 |
| Klose..... | Mehlnährschaden and edema | 1¼ | 3,031 | 2,619 | 412 | 1.3 | 1.58 | 9.4 | 11.6 |
| Steinitz..... | Atrophy | 3 | 2,625 | 2,098 | 527 | 1.5 | 2.52 | 7.2 | 11.6 |

* Collected in part by Murlin and Hoobler: Energy Metabolism of Ten Hospital Children Between the Ages of Two Months and One Year, Am. J. Dis. Child. **9**: 81 (Feb.) 1915.
† Average of six infants.

body substance were uniform despite the different degrees of undernutrition (excluding the subjects with edema). The importance of these results will be considered in the following section.

The following possibilities have been suggested to explain the changed level of the basal metabolism which exists in the infants who are 30 per cent or more underweight: (1) The proportion of active metabolic to inert metabolic tissue may remain uniform in all degrees of emaciation (over and above a weight loss of 30 per cent), or (2) the proportion of active to inert tissue may change with progressive emaciation.

In either case, the production of heat per kilogram of active metabolic tissue may be higher, lower or the same in this condition as during health.

As previously mentioned, the analyses of the composition of the bodies of the atrophic infants [20] showed a uniform relationship between nitrogenous material (active protoplasmic tissue) and body weight in all grades of malnutrition. On the basis of these results, it would seem reasonable to eliminate from consideration the second possibility, at least until further evidence is forthcoming. This comment, therefore, will be limited to a consideration of the first group of possibilities in which the proportion of active to inert tissue is assumed to remain uniform in infants who are more than 30 per cent underweight. The reduction in height which usually accompanies the loss of weight in these infants also supports this view.

Many workers are of the opinion that the atrophic infant may, under different conditions, produce more, less or the same amount of heat per kilogram of active tissue as the normal infant. For example, Talbot stated [22] that "it is reasonable to conclude that as long as the body is able, it responds to the demand (increased heat loss) put on it and produces extra heat." He believed, however, that in one group of marantic infants "the body fat merely serves as a diluent of the metabolism," whereas in another group "the heat forming organs are incapable of forming the same amount of heat as during health." Similarly, Fleming [6] also ascribed differences of metabolism that had been observed in moderate and severe malnutrition to a higher production of heat per kilogram of active tissue in the one instance and to a lower production of heat in the other instance.

The basal figures of all the marantic infants who were more than 30 per cent underweight as reported by the foregoing observers,[23] as well as the figures for the present group of infants, were examined in an effort to throw light on this disputed question. The technic and conditions of the experiments were uniform and the results, therefore, comparable. In table 5, these observations are arranged to show the severity of the malnutrition, expressed as percentage of expected weight, and the output of heat per kilogram of true weight.

If the body were stimulated to produce more heart in response to an increased loss of heat, the heat output per kilogram of metabolically active tissue and, consequently, of total body weight would theoretically rise progressively as the emaciation became more marked. It would presumably reach a maximum in the most extremely atrophic infants who are almost entirely devoid of subcutaneous fat,[24] provided, of

22. Talbot (footnote 5, first reference, p. 369).

23. Fleming and Hutchison (footnote 2). Murlin and Hoobler (footnote 4). Talbot (footnote 5, first reference). Fleming (footnote 6).

24. Klose, E.: Zur Kenntnis der Koperzusammensetzung bei Ernahrungsstorungen, Jahrb. f. Kinderh. **91**:157, 1920.

TABLE 5.—*The Relation Between the Degree of Emaciation and the Basal Metabolism in Marasmus (from the Literature and the Present Study)*

| Percentage of Expected Weight for Age | Weight, Kg. | Calories | | Reference and Case * |
|---|---|---|---|---|
| | | Total | Kg. per 24 Hours | |
| 31 | 2.49 | 185 | 74 | Fleming and Hutchison—Can. |
| 36 | 2.25 | 174 | 77 | Levine and Wilson—7 |
| 37 | 3.62 | 313 | 87 | Talbot (new series)—11 |
| 38 | 3.68 | 324 | 88 | Fleming and Hutchison—By. |
| 38 | 2.63 | 193 | 73 | Talbot (new series)—8 |
| Average | | | 80 | |
| 40 | 3.49 | 282 | 81 | Levine and Wilson—4 |
| 41 | 2.49 | 176 | 71 | Murlin and Hoobler—C. P. |
| 41 | 3.21 | 284 | 88 | Fleming (average of 3 experiments)—Cn. |
| 42 | 3.38 | 297 | 88 | Benedict and Talbot—J. V. |
| 43† | 3.33 | 345 | 104 | Talbot (new series)—14 |
| 47 | 3.54 | 302 | 85 | Fleming (average of 5 experiments)—Be. |
| 47 | 3.37 | 260 | 77 | Fleming—McN. |
| 48 | 2.94 | 242 | 81 | Levine and Wilson—3 |
| 49 | 2.68 | 228 | 85 | Fleming and Hutchison—An. |
| 49† | 2.35 | 246 | 108 | Talbot (new series)—13 |
| Average | | | 82 | |
| 50 | 3.60 | 272 | 76 | Talbot (new series)—17 |
| 50† | 2.60 | 286 | 110 | Talbot (new series)—3 |
| 50 | 3.80 | 325 | 86 | Fleming—McD. |
| 51 | 3.41 | 246 | 72 | Murlin and Hoobler—C. D. |
| 51 | 3.18 | 226 | 71 | Benedict and Talbot—A. L. |
| 51 | 2.69 | 194 | 72 | Levine and Wilson—10 |
| 51 | 4.13 | 376 | 91 | Fleming—Wt. |
| 52 | 4.70 | 370 | 79 | Levine and Wilson—8 |
| 52 | 4.00 | 313 | 78 | Fleming (average of 6 experiments)—Ls. |
| 52 | 2.63 | 167 | 63 | Fleming and Hutchison (average of 2 experiments)—Ce. |
| 52 | 3.82 | 345 | 90 | Fleming and Hutchison—Ln. |
| 53 | 3.71 | 274 | 74 | Levine and Wilson—5 |
| 53 | 4.48 | 403 | 90 | Fleming (average of 4 experiments)—McG. |
| 54 | 4.28 | 326 | 76 | Fleming (average of 3 experiments)—Ry. |
| 55 | 3.74 | 290 | 78 | Fleming—Stn. |
| 55 | 3.31 | 234 | 71 | Fleming—Pr. |
| 56 | 5.18 | 369 | 71 | Benedict and Talbot—D. M. |
| 57† | 3.48 | 171 | 49 | Fleming—Sn. |
| 58 | 4.53 | 380 | 83 | Fleming and Hutchison—Nn. |
| 58 | 3.71 | 284 | 76 | Fleming—Le. |
| 58 | 4.65 | 348 | 75 | Fleming—Fy. |
| 59† | 3.70 | 407 | 110 | Talbot (new series)—12 |
| Average | | | 81 | |
| 60 | 2.05 | 169 | 82 | Fleming and Hutchison (average of 2 experiments)—Sl. |
| 60 | 4.60 | 328 | 71 | Talbot (new series)—7 |
| 60 | 5.76 | 434 | 74 | Murlin and Hoobler—J. Sm. |
| 61 | 4.00 | 374 | 93 | Talbot (new series)—4 |
| 62 | 3.96 | 319 | 80 | Fleming and Hutchison—Se. |
| 63 | 4.30 | 392 | 91 | Talbot (new series)—15 |
| 63 | 3.74 | 282 | 76 | Fleming (average of 2 experiments)—Me. |
| 64 | 3.97 | 267 | 67 | Fleming and Hutchison (average of 2 experiments)—Ste. |
| 65 | 6.00 | 405 | 68 | Talbot (new series)—10 |
| 65 | 6.88 | 504 | 73 | Fleming and Hutchison—By. |
| 66 | 5.17 | 378 | 73 | Levine and Wilson—5 |
| 66 | 5.63 | 467 | 83 | Benedict and Talbot—J. M. |
| 66 | 5.00 | 406 | 81 | Fleming—Cl. |
| 66 | 5.35 | 406 | 76 | Fleming and Hutchison—Cn. |
| 67 | 3.76 | 321 | 85 | Fleming and Hutchison—Bn. |
| 67 | 5.45 | 387 | 70 | Benedict and Talbot—J. P. |
| 67 | 5.84 | 443 | 76 | Talbot (new series)—6 |
| Average | | | 79 | |

* Only those observations are included in which the infants slept throughout or were awake for short intervals but were quiet.

† These observations are not included in the averages.

course, that complicating factors such as excessive wasting of active tissue did not supervene.[25]

An analysis of the results (table 5) does not support this view. A striking uniformity of the basal rate was found to exist in all of the infants. The average production of heat of the markedly emaciated infants who were as much as from 60 to 70 per cent underweight and of the moderately malnourished infants who were only from 30 to 40 per cent underweight was practically identical (80 and 79 calories per kilogram per twenty-four hours, respectively). Similarly, the intermediate groups of infants who were from 40 to 50 and from 50 to 60 per cent underweight produced 82 and 81 calories per kilogram per

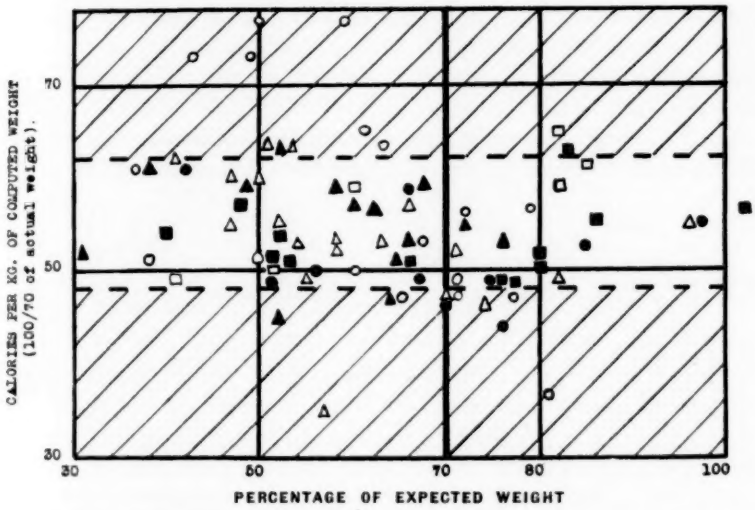


Chart 2.—The degree of malnutrition expressed as percentage of expected weight (for age), and the basal metabolism per kilogram of computed weight (100/70 of the actual weight) of marantic infants as reported in the literature and in the present study. The results in infants who are less than 30 per cent underweight are included for comparison. The area within the broken lines shows the normal range of the basal metabolism in infancy. The white circles represent the cases of Talbot (new series); the black circles, those of Benedict and Talbot; the white triangles, those of Fleming; the black triangles, those of Fleming and Hutchison; the white squares, those of Murlin and Hoobler and the black squares, those of the present series.

twenty-four hours. Deviations from the average figure of 80 calories per kilogram exceeded ± 15 per cent in only eight of the fifty-four observations.

25. The data on the composition of the infant's body, previously mentioned, is against this view.

The evidence seems adequate to establish the basal rate of emaciated infants devoid of body fat at the high level of approximately 80 calories per kilogram per twenty-four hours as against a basal rate of 55 calories (± 10 per cent) for well-nourished infants with normal reserves of fat. This inert tissue which may, according to Fleming and others,[26] comprise as much as 35 per cent of the total body of the latter infants, presumably does not exert any influence on metabolism. The production of heat of the marantic infants recorded in table 4 was therefore calculated on the basis of this additional weight (100/70 of the actual weight). The results together with the degree of emaciation, expressed as percentage of expected weight, are shown in table 6 and in chart 2. The table gives only the average values for differences in weight of 10 per cent; the chart gives the results for individual infants. The values for infants who are less than 30 per cent underweight are included for comparative purposes.

TABLE 6.—*The Relation Between the Degree of Emaciation and the Basal Metabolism per Kilogram of Computed Weight (100/70 of Actual Weight) from the Literature and the Present Study*

| Percentage of Expected Weight for Age | Number of Observations | Calories per Kg. of Computed Weight (100/70 of Actual Weight) Average |
|---|---|---|
| 30 to 39 | 5 | 56 |
| 40 to 49 | 8 | 58 |
| 50 to 59 | 19 | 54 |
| 60 to 69 | 17 | 55 |
| 70 to 79 | 16 | 60 |
| 80 to 89 | 8 | 57 |
| 90 to 99 | 3 | 56 |

When the absence of inert tissue is taken into account, the basal rate is the same in all degrees of marasmus as in health. The output of heat of only thirteen of the fifty-four infants who were more than 30 per cent underweight was outside the limits of the normal range, and in only seven of these infants was the deviation significant.

It seems justifiable to conclude from this evidence that the differences in the basal metabolic rate of well nourished, undernourished and emaciated infants depend solely on the presence in the first group of infants of metabolically inert tissue which constitutes approximately 30 per cent of the total body weight and not on any alteration in the character of the fundamental metabolism. The active protoplasmic tissue per kilogram apparently produces the same amount of heat in marasmus as in health.

The normal production of heat per kilogram of active tissue which is found even in marked emaciation indicates that the tendency of these

26. Fleming (footnote 6). Tisdall, Drake and Brown (footnote 9).

infants to lose abnormally large amounts of heat by radiation and conduction as a result of the absence of subcutaneous fat is counteracted chiefly by contraction of superficial capillaries and diminution of blood flow (physical regulation of body temperature). An increased production of heat (chemical regulation) probably played a rôle in maintaining a normal body temperature only in a few infants whose basal rate was inordinately high (from 104 to 110 calories per kilogram), as shown in table 4.

In the preceding comment, the hypothesis was tentatively advanced that the presence of relatively different amounts of metabolically inert tissue in the bodies of well nourished, undernourished and emaciated infants explains the different levels of the basal metabolism which are found in all stages of nutrition. This conception explains the facts simply and satisfactorily on a single basis and obviates the necessity of assuming abnormal metabolic processes. The possibility remains, however, that a combination of interrelated factors, some increasing and some decreasing production of heat, may operate in such a manner as to produce the same end-results. In the absence of reliable evidence demonstrating such factors, the former view seems justifiable, for the present.

## SUMMARY

The basal metabolism of four normal and of six marantic infants was determined in fourteen observations of from one to three hours' duration with the aid of a respiration chamber apparatus. One infant was studied at intervals during which his weight steadily increased from 53 to 80 per cent of that expected for his age.

The basal rate of all the control infants, which ranged from 51 to 63 calories per kilogram per twenty-four hours, closely approximated the standard figures of Benedict and Talbot.[3]

The basal rate of the markedly marantic infants was invariably high per kilogram of actual weight (from 72 to 82 calories) and invariably low per kilogram of expected weight (from 28 to 41 calories). The basal rate of the infant whose weight increased from 53 to 66 per cent of the normal weight rose from the initial low level of 39 calories per kilogram of expected weight to the lower limit of the normal range of 48 calories per kilogram. When his weight increased further to exceed 75 per cent of the normal weight, his basal rate on the basis of actual weight also approached the upper limit of the normal range in three observations (64 calories per kilogram).

## CONCLUSIONS

The evidence in the literature and in the present study establishes the basal metabolic rate of marantic infants devoid of deposits of fat at the high level of about 80 calories per kilogram per twenty-four hours

as against the basal rate of 55 calories (± 10 per cent) for well nourished infants with normal fat reserves.

The explanation for the changed level of the basal metabolism in infantile marasmus is not yet certain. Evidence of abnormal metabolic processes occurring in this condition is lacking. Evidence has been presented which suggests that the changed level of the basal metabolism of the marantic infant may be due to the marked diminution in its organism of metabolically inert tissue which comprises about 30 per cent of the body of the well nourished infant. The character of the fundamental metabolism apparently remains normal in marasmus, and the production of heat per kilogram of active protoplasmic tissue appears to be essentially the same in this condition as in health.

161 West Sixty-First Street.

# Case Reports

## PERICARDITIS AS A PRIMARY CLINICAL MANIFESTATION OF TUBERCULOSIS IN CHILDHOOD *

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Pericarditis as the first clinical manifestation of active tuberculosis in childhood is rare. A careful review of the literature failed to reveal the report of such a case diagnosed during life.

Waller [1] reported a case in which postmortem examination showed that the pericarditis was tuberculous; tuberculous pericarditis as a postmortem observation is not a great rarity.[2] Many cases of chronic phthisis reveal an adhesive pericarditis at autopsy, and in generalized miliary tuberculosis the pericardium is not infrequently involved along with other serous membranes. Norris [3] collected data on 1,780 cases of tuberculosis from various hospitals; eighty-two cases of tuberculous pericarditis were found, thirty-two in which there were actual tubercles, and forty-nine in which tubercles or any other demonstrable etiology was not found. Machlachlan [4] reviewed 975 autopsies in which 100 cases of pericardial involvement of one type or another were included; ten per cent of these were on a tuberculous basis. Wells [5] made a study of 1,048 autopsies made on adults with a view to determine the pathogenesis of healed fibrous adhesions of the pericardium. He found inflammatory changes in the pericardium in 128, while fifty-seven presented chronic inflammatory changes which had resulted in fibrous adhesions joining the two layers. The remaining seventy-one of the number examined had had an acute condition. In eight patients the condition had been of the chronic type on a rheumatic basis; in six,

* Received for publication, Nov. 21, 1927.

1. Waller, W. E.: Tuberculosis of Pericardium, Lancet **2**:278 (Aug. 2) 1923.
2. Crawford, B. L.: Proc. Path. Soc., Philadelphia, vol. 23, p. 82. Rawls, W. B.: Am. J. M. Sc., vol. 169, p. 875. Jauch, J. F.: Brit. M. J. **1**:798, 1922.
3. Norris, W.: Cardiac Pathology, Philadelphia, 1911, p. 113.
4. Machlachlan, W. W. G.: Am. J. M. Sc., vol. 162, p. 654.
5. Wells, H. G.: Am. J. M. Sc., vol. 123, p. 241.

the pericarditis was accompanied by frankly tuberculous lesions, such as caseous masses or tubercles, and forty-three cases were of undeterminable etiology. Wells felt that many of these cases of chronic adhesive pericarditis of unknown etiology were on a tuberculous basis, the inflammatory change occurring not as a direct action of the tubercle bacillus on the serous membrane, but as a result of the action of tuberculous toxin liberated by some focus elsewhere in the body.

In discussing tuberculous pericarditis in childhood, Bass [6] reported this condition to be less common in children than in adults. He quoted an early writer who found only eighteen cases of tuberculous pericarditis in children under 15 years of age in a series of fifty-one cases. Fischer [7] stated that tuberculosis of the pericardium in childhood is rarely met with as a primary process but that it usually occurs as a part of a generalized miliary dissemination in which all organs of the body are involved. He further stated that a positive diagnosis will probably be made only at necropsy.

## PATHOGENESIS

Infection of the pericardium may occur in any one of a variety of ways. Primary tuberculous pericardiditis, in the strict sense of the word, is exceedingly rare; the disease is usually secondary to some focus elsewhere in the body. Infection may occur by the blood stream as a part of a generalized miliary distribution, by the lymphatics, by direct extension from an inflamed mass of hilum glands or pleura, or rarely, by rupture of a caseating hilum gland into the pericardial sac. Tuberculous pericarditis seems to bear a more intimate relationship to involvement of the hilum gland than to foci of tuberculosis elsewhere in the body.[4, 8] Infection occurs most commonly through the lymphatics or by direct extension from these glands. In generalized miliary tuberculosis, the pericardium is involved less frequently than other serous membranes. The type of case in which an acute pericarditis is the initial manifestation of the disease is not associated with a widespread tuberculosis, as a rule.[4]

## PATHOLOGY

The essential pathologic changes vary with the pathogenesis. When the pericardial involvement occurs as a part of a generalized miliary distribution, marked changes are not commonly noted. The serous surface is roughened by relatively few scattered tubercles, and there is a moderate increase in the pericardial fluid.

The more chronic types are characterized by large amounts of exudate, which may be fibrinous, serofibrinous or hemorrhagic. When

6. Bass, M. H.: Abt's Pediatrics **4**:344, 1925.

7. Fischer, Louis: Diseases of Infants and Children, Philadelphia, 1925, p. 391.

8. Rigler, L. G.: Journal-Lancet, vol. 64, p. 155.

large fibrinous deposits are present, scattered tubercles may occur, and deep within the mass, caseation is occasionally found.

In most instances, fluid effusions constitute the predominant portion of the exudate, and the amount of effusion in this type of pericarditis is apt to be larger than in any other, often yielding 2 liters or more.[9] The effusion is most often serous, though it may be serosanguineous or even purulent.

As the process continues, adhesions appear between the parietal and visceral pericardia, resulting in trabeculations with accumulations of fluid in the intervening spaces. The exudate is absorbed very slowly, and in long continued cases, the pericardial sac may be completely obliterated. Rarely, calcification of the pericardium may occur. Secondary infection of other serous cavities with a resultant generalized serositis, presenting the picture of Pick's disease or a generalized miliary involvement, may occur as a terminal condition; both conditions existed in our case.

### SYMPTOMATOLOGY

Primary tuberculous pericarditis is characterized at the onset by a syndrome closely resembling that of an acute nontuberculous pericarditis. The onset is abrupt, with pain over the precordium, high temperature, dyspnea and prostration. Physical examination early reveals a loud rub over the precordium, with possibly some increase in cardiac dulness and usually a cutaneous hypersensitiveness. As the disease progresses, physical signs of pericardial effusion make their appearance. The amount of fluid increases more or less rapidly, until the effusion becomes enormous, giving all the classic physical signs. The course of the disease is characterized by its chronicity, the effusion resorbing very slowly. As a rule, great cardiac embarrassment results, and frequent tappings are necessary for relief.

### DIAGNOSIS

Tuberculous pericarditis with effusion must be considered when the amount of fluid is excessive, for large amounts of fluid characterize this condition. The fluid is more frequently sanguineous in tuberculous than in rheumatic pericarditis, and it refills the sac with startling rapidity after removal. The specific gravity is that of an exudate around 1.020 rather than the lower figures of a transudate. Tuberculin skin reactions indicating the presence of a tuberculous infection are extremely helpful in making a diagnosis. Culture, microscopic search and finally inoculation of guinea-pigs give the conclusive evidence while the patient is alive. The prognosis is always unfavorable, the condition terminating in death or in a chronic adhesive pericarditis.

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9. Wilson, F. N., and Hills, C. R.: M. Clin. N. Amer. **6**:1201, 1919.

### THERAPY

The usual general hygienic measures employed in the treatment of a patient with active tuberculosis are indicated.

Paracentesis of the pericardium is a valuable procedure in these cases, since it not only relieves cardiac embarrassment, but seems to hasten the absorption of the exudate. It is of great value as a diagnostic procedure as well, for while it is difficult to find the organism in the exudate, inoculations of guinea-pig usually give positive results. Injection of air into the pericardial sac has also been recommended (Wenckebach) as a therapeutic measure.

The technic of pericardial paracentesis merits discussion. Several areas have been used as the site of puncture. The most satisfactory method from the point of view of ease of execution and safety, in our experience, is the following:

A point is selected half way between the left outer border of dulness and the apex beat in the fifth interspace. An 18-20 gauge 4 inch (132.1 cm.) needle bearing a stylet is most satisfactory. The needle is introduced into the wall of the chest, pointing slightly upward and medially. As the pleura is met, a feeling of resistance is encountered. When the parietal pericardium is encountered, a second sense of resistance is noted. When the second membrane is pierced, the needle lies in the pericardial cavity. If it is released, it will be found to pulsate in a large arc synchronously with the cardiac cycle. The stylet is then withdrawn and the fluid collected.

It is often necessary to puncture the pericardium successively at more than one point as the condition becomes chronic and the fluid becomes pocketed. Right-sided puncture is as a rule a little more dangerous. The right ventricle is much thinner than the left, and in the presence of a right-sided dilatation, a not uncommon occurrence, the needle may slip through the muscular wall and into the ventricle without the operator's knowledge. While this is not especially serious, it is not pleasant to puncture the heart, and it is apt to cause distress to the patient. Fluid is drawn off as completely as possible, only the force of gravity being used or aspiration with a syringe. Other more forceful methods of aspiration should be avoided, as they are dangerous because the heart itself may be injured.

### REPORT OF A CASE

*History.*—Walter H., colored, aged 9, was admitted to the Children's Ward, Cook County Hospital, April 27, 1925. He had been in good health until two weeks previously, when he had begun to cough. A week later, he had complained of headache and had had a slight rise in temperature in the evening. Thirty-six hours before admission, he was seized with sharp, knifelike constant pain in the left side of the chest, which was aggravated by respiration.

His previous illnesses were mumps, measles and colds. He had never had a severe sore throat, chorea or other rheumatic manifestations. History of contact with tuberculosis was not obtainable.

*Examination.*—Examination revealed a well nourished, acutely ill child, with a temperature of 102 F., a pulse rate of 98, and respiration, 24. The chest showed compression consolidation of the lower lobe of the left lung. Other lung observations were not noted. The apex beat was visible diffusely over the fourth and fifth interspaces, just within the nipple line. The right border of the heart was 1 inch (2.5 cm.) to the right of the right sternal margin in the fourth interspace and one-half inch (1.27 cm.) in the second interspace; the left border was just outside the anterior axillary line. A loud friction rub was heard over the precordium. The liver and spleen were not palpable.

Examination of the blood on admission showed: hemoglobin, 85 per cent; red blood cells, 5,200,000, and white blood cells, 13,500. The results of urinalysis were negative.

A diagnosis was made of pericarditis with effusion of questionable type.

*Course of Illness.*—On April 30, the friction rub was diminished, and the heart tones were distant. Fifty cubic centimeters of a blood-tinged fluid was withdrawn from the pericardium; it had a specific gravity of 1.020 and contained albumin and numerous red cells. Organisms were not found on the smear. Cultures of this fluid and all subsequent specimens were sterile; smears for tubercle bacilli were constantly negative. At this time, a diagnosis of tuberculous pericarditis was suggested, and later confirmed by a positive Pirquet test and positive results from inoculation of guinea-pigs.

Subsequently the child had an elevated temperature continuously; it was remittent, ranging from 99 to 100 F. in the morning to 103 to 104 F. in the evening. Until July 18, there was marked dyspnea necessitating pericardial tapping at weekly intervals. The amount of fluid removed varied from 100 to 850 cc., and the blood in the fluid gradually diminished. On July 12, the exudate was a clear amber and remained so. Clinical improvement dated from this time, and on July 18 the last tapping resulted in 350 cc. of a clear amber fluid. The total amount of fluid withdrawn during the ten punctures amounted to 5,130 cc. The patient was discharged from the hospital on September 9, with an enlarged heart with distant heart tones but no murmurs or friction rubs.

Roentgen-ray results corroborated the size of the pericardial effusion, showing slight hilum shadows and no other abnormalities in the lungs.

*Admission to Municipal Tuberculosis Sanitarium.*—His subsequent history was obtained from the social service department of the Cook County Hospital, and Dr. K. J. Hendrickson, of the Chicago Municipal Tuberculosis Sanitarium. He was hospitalized at St. Luke's Hospital on Jan. 7, 1926, and discharged from there to the Municipal Tuberculosis Sanitarium in April, where he died on June 7.

Examination on admission to the sanitarium revealed an emaciated child with enlarged supraclavicular glands, an enlarged heart with distant tones but without murmurs. There was impaired resonance over the bases of both lungs posteriorly. The abdomen was much distended, and fluid was demonstrated.

Roentgen-ray examination by Dr. C. E. Cook suggested pericarditis with effusion and fluid in the right side of the chest cavity.

The sputum was negative for tubercle bacilli.

*Autopsy.*—Autopsy was performed June 8, 1926, by Dr. H. C. Sweany and Dr. M. Wasick. The anatomic diagnosis was as follows: (1) adhesive tuberculous peritonitis; (2) generalized serositis with fibrinous deposits in the peritoneal, pericardial and pleural cavities; (3) tuberculous tracheobronchial, tracheal, retroperitoneal and mesenteric lymphadenitis; (4) miliary tuberculosis of the lungs, liver and kidneys and (5) passive congestion of the lungs, liver, spleen and kidneys.

## COMMENT

This case illustrates the course which may be followed by an acute tuberculous pericarditis. Prior to the pericarditis, the child apparently had an unrecognizable tracheobronchial tuberculosis. As long as the bacilli remained confined to this region, he did not show marked symptomatology, but as soon as the pericardium was involved, he suddenly became seriously ill. That the pericarditis was the initial symptom in this case is beyond question. As time went on, the infection in the pericardial sac gradually subsided and finally healed, yielding an adhesive pericarditis. Subsequent to the patient's discharge from Cook County Hospital, his symptoms were never again referable to the pericardium. While at St. Luke's Hospital and at the sanitarium, the predominant observations were abdominal, the pericarditis being noted merely incidentally in a roentgen-ray examination. Some time subsequent to his discharge from the county hospital, therefore, other serous membranes were involved, resulting in the polyserositis observed on his admission to the sanitarium. As the final stage in the disease, a generalized miliary distribution occurred.

## CONCLUSIONS

1. An acute pericarditis may be the initial manifestation of a tuberculous infection.

2. The clinical picture in these cases is fairly clear cut.

3. Laboratory procedures, as tuberculous skin reactions and guinea-pig inoculation tests, are of great value as diagnostic agents.

4. Pathologically, the sequence of events is definite, yielding an obliterative pericarditis and not infrequently a polyserositis or a generalized miliary dissemination.

5. Prognostically, these cases belong to the malignant class of tuberculous infections, similar in nature to meningeal involvement.

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# CHYLANGIOMA OF MESENTERY IN A CHILD*

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A case of chylangioma of the mesentery in a child is reported because of its rarity.

*History.*—J. H., the patient, was a white American boy, 13 years of age. The mother died during her seventh pregnancy, the cause of death not being known. The father died of rheumatism and chronic interstitial nephritis with marked dropsy. Otherwise the family history was uninteresting. A history of the mother's pregnancies is as follows: The result of the first pregnancy was a girl who at the time this history was taken was 24 years old and well; the second child was a boy, aged 23, and well; the third, a girl, aged 20, and well; the fourth child was the patient. The fifth and sixth pregnancies resulted in miscarriages. The mother died during the seventh pregnancy.

The patient was born at full term; delivery was normal, and a physician was in attendance. The patient was said to have been a large baby and to have been breast fed until he was 9 months old. He had had measles, and whooping cough, and had been treated in a hospital for appendicitis, but an operation had not been performed. His eating habits were irregular, but his food seemed to be well balanced. He was constipated, and had used many drugs for this condition. The child talked and groaned in his sleep. The history was exceedingly unsatisfactory, having been obtained from a relative who knew little about the early life of the child.

For four years before the present illness, the patient had been complaining of pain in the right side of the abdomen, and there had been soreness to the touch at this point. When he came to live with his sister three and a half years ago, there was a lump the size of a hen's egg, in the right side of the abdomen just below the waist, and there had been periodic attacks of pain without nausea. During the patient's residence in another state, he is said to have been treated for chronic appendicitis and also to have been treated with the roentgen ray for the knot.

*Physical Examination.*—His height was 59 inches, his weight 84 pounds and 12 ounces (28 Kg.); average weight was 90 pounds (40 Kg.). The general appearance was that of a fairly well developed and well nourished boy, who was not acutely ill. His mentality was good. A few small lymph nodes were felt in the neck and one epitrochlear on the left side. Inspection of the abdomen showed a small mass midway between the umbilicus and the anterior superior spine on the right side; it did not move with respiration. On palpation, which was somewhat difficult because the patient was unable to relax, a firm, irregular mass was found which was somewhat tender and not freely movable. It appeared to be within the abdominal cavity and apparently attached somewhere near the midline of the body. This mass could not be palpated on rectal examination.

* Received for publication, Jan. 20, 1928.

* From King Daughter's Clinic, Norfolk, Va.

*Laboratory Examination.*—Urinalysis was negative. Examination of the blood showed: 6,500 white blood cells, differential count normal and 4,500,000 red blood cells; the red cells varied slightly in size. The results of the examination otherwise were normal.

Consultation was held with the attending surgeon, but a diagnosis could not be arrived at. An exploratory operation was deemed advisable. Two days later, an operation was performed. A tumor 10 or 12 cm. in diameter was

Fig. 1.—Gross specimen as removed. The photograph, unfortunately, was not taken until the tumor had been cut into and a block removed for frozen section.

found in the mesentery of the small intestine, closely adjacent to, but not involving, the intestine. The blood supply of the intestine was so much involved from the tumor that a resection of the intestine was necessary in order to remove the tumor. A specimen consisting of mesentery with a loop of small intestine about 44 cm. in length on one side of the tumor and 14 cm. in length on the other side, was sent to the laboratory. An indefinitely outlined mass occupied the mesentery and involved its wall on one side. The mass had a grayish, roughened external surface with raised yellowish nodules scattered

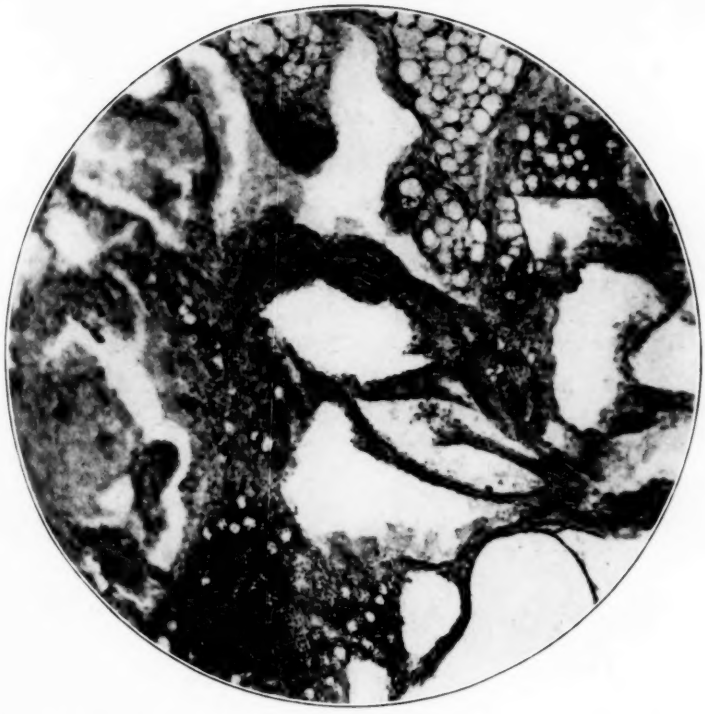

Fig. 2.—Permanently fixed and stained section; magnified 60 diameters.

Fig. 3.—Same as figure 2, magnified 150 diameters.

over the surface. On section through the center of the mass, the cut surface was dark red, of spongy consistency, and showed many dilated and cystic vessels, from which milky fluid, slightly blood tinged, could be expressed. The appendix was 16 cm. long, the external surface was smooth and the mesoappendix very fat. The lumen of the appendix was filled near the distal end with fecal masses. On section, the mucosa appeared to be normal for about 2 cm. from the proximal end; from this point to the distal end, there was marked hypertrophy, the mucosa being much congested and covered by hemorrhagic exudate. Frozen sections of the tumor showed dilated lacteals and lymph spaces. The surrounding tissue showed simple hyperplasia of lymph nodes.

The specimen was fixed. Gross examination revealed a tumor about 10 or 12 cm. in diameter in the mesentery of the small intestine, adjacent to the intestine but not invading it. The tumor was somewhat spongy in consistency, and for the most part was almost black like old blood.

On microscopic examination, sections of tumor showed numerous dilated lymph vessels, many of which were filled with blood, but some with lymph only; numerous large lymph nodes with marked hyperplasia, and many diffuse lymphoid collections.

The diagnosis was lymphangioma (chyloma) of the mesentery and marked hyperplasia of the lymph nodes.

# CONGENITAL NONROTATION OF THE STOMACH WITH EVENTRATION *

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AND
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Eventration of the diaphragm is a relatively rare condition and almost always occurs on the left side. In 1916, Bayne-Jones [1] reviewed forty-five cases of eventration, the total number of cases reported up to that time. In only two of the forty-five cases did eventration occur on the right side. The first case in the literature was reported by Wieting.[2] The patient was a man, aged 50, and the eventration was probably congenital. In 1911, Eppinger [3] reported a case with typical signs of eventration in a man, aged 35; in this case, too, the condition was congenital and was not seen during life. In both cases, there were no other associated anomalies. Bayne-Jones [1] reported the third case in a man, aged 52. Jones' patient had no symptoms except constipation and indefinite pain in the upper part of the right side of the abdomen. Jones considered his case the first one of eventration of the right side in which an intra vitam diagnosis was made. The diagnosis was confirmed by operation. In his case, the diaphragm reached the level of the third rib and showed paradoxic movements, that is, the diaphragm ascended with inspiration and descended with expiration. The liver was high up and was immediately below the diaphragm. Except that it was in a high position, the stomach was not displaced. The pylorus was found under the right costal margin. When first seen, the patient presented the signs found in thickened pleura and in fluid in the pleural cavity. Roentgen-ray examination revealed the true diagnosis. In 1927, Levick [4] reported a case of eventration on the right side in a girl, aged 5 years. Levick stated that his was the first case reported in the British literature, and he considered it identical, clinically and roentgenologically, with the case described by Bayne-Jones.

* Received for publication, Jan. 17, 1928.

1. Bayne-Jones: Eventration of Diaphragm, Arch. Int. Med. **17**:221 (Feb.) 1916.

2. Wieting: Deutsche Ztschr. f. Chir. **82**:314, 1903.

3. Eppinger: Allg. u. Spez. Path. der Zwerchfells, suppl. Spez. Path. u. Therap., 1911.

4. Levick, C. B.: Eventratio diaphragmatica, Proc. Roy. Soc. Med. **20**:51 (July) 1927.

In 1921, Kinney [5] reported a case of incomplete embryonic diaphragmatic hernia with congenital nonrotation of the stomach in a female infant, 16 months of age. The chief complaint was violent dysphagia accompanied by paroxysms of coughing and vomiting. Roentgen-ray examination revealed the diaphragm high up and the stomach above the liver on the right side in its proximal two-thirds, the distal one-third lying below the liver. These two parts communicated through what would be the normal position of the esophagus. The pylorus was found in the midline, tucked up against the liver. The stomach emptied itself in six hours. The cecum was found in the middle of the hepatic margin, and the ascending colon followed the margin in the usual nonrotated, nondescending position. Although Kinney classified his case as one of incomplete embryonic hernia, according to the classification of Paillard,[6] it was undoubtedly a case of congenital eventration of the diaphragm with nonrotation of the stomach. Paillard's original classification is as follows:

1. Embryonal diaphragmatic hernia
   *A.* Complete—the entire absence of the pillars of Uskow
   *B.* Incomplete—partial absence of the pillars of Uskow
2. Fetal hernia, due to local arrested development or to insufficient development of part of the diaphragm

In other words, a defect in the diaphragm must be present if a case is to be classified as incomplete diaphragmatic hernia. Apparently, this defect was not found in Kinney's case, and therefore, it must be designated as one of eventration. Recently, Harris and Stivelman [7] reported a case of nonrotation of the stomach with eventration of the right side of the diaphragm simulating spontaneous hydropneumothorax in a youth, aged 22.

Glässner [8] and Eppinger [9] have described cases of eventration of the right side of the diaphragm; in both cases the stomach was rotated to 180 degrees. Roentgen-ray examination showed a large, air-containing space above the liver, which was demarcated from the lung field by a sharp line.

It is evident that of the eight cases of eventration of the right side of the diaphragm thus far reported, four occurred with, and four with-

5. Kinney: Congenital Nonrotation of Stomach, Am. J. Roentgenol. **8**:383 (July) 1921.

6. Paillard: Les Varietés anatomiques de la hernie diaphragmatique congénital, Faculté de medicin de Paris, 1903.

7. Harris and Stivelman: Nonrotation of Stomach Simulating Spontaneous Hydropneumothorax, J. A. M. A. **89**:1836 (Nov. 26) 1927.

8. Glässner, cited by Assman: Forschung der Roentgenologie, vol. 24.

9. Eppinger: Zur Roentgen diagnostik und pathologischen Anatomie einer Hernia Diaphragmatica paraesophagea, Ztschr. f. Heilk., 1904, vol. 11.

out, an abnormal position of the stomach. Only one of these cases occurred in an infant (Kinney [5]).

The case reported here represents the fifth case of true eventration with abnormal position of the stomach.

## REPORT OF A CASE

*History.*—P. C., a girl (referred by Dr. B. Kaufman), was first seen on Oct. 20, 1925, when 5 weeks of age. She had been born at full term and the delivery was normal. She was the fourth child, and at birth weighed 9 pounds (4.1 Kg.). The baby was breast fed, but the mother did not make an attempt to maintain a regular feeding schedule because the infant did not take the breast well and cried throughout each nursing period. The infant was usually kept at the breast for ten minutes. Vomiting was absent. The stools were frequent and loose, and they contained mucus and curds. The mother also said that the infant was very restless.

*Physical Examination.*—Poor turgor and poor muscle tone were revealed. The weight was 9 pounds, 2 ounces. The anterior fontanel was 4 by 2.5 cm. The posterior fontanel was still open; otherwise, results of the physical examination were negative.

The infant was seen at regular intervals from Oct. 20, 1925, until Jan. 21, 1926. During this time the appetite remained poor, vomiting did not occur and slow gains in weight were noted. On January 21, the mother said that the infant had been exposed to whooping cough. Four prophylactic injections of 1 cc. each of pertussis vaccine (New York Health Department) were given without benefit; in spite of the injections, the child contracted a severe case of whooping cough; the paroxysms were frequent and severe, coming on at ten minute intervals. On April 6, the infant was brought for examination because it had vomited almost everything taken for the past four or five days; it weighed 13 pounds, 2 ounces (6 Kg.) and had practically recovered from its attack of whooping cough. The color and turgor were poor; otherwise, physical examination did not reveal anything abnormal. The vomiting persisted until April 20. Repeated attacks of vomiting occurred at irregular intervals until Aug. 20, 1927, when the infant was again brought for examination because of vomiting which had been present for a few days. Vomiting was profuse and recurred from four to five times a day. The weight was 20 pounds, 1 ounce (9 Kg.); the temperature was normal, although the mother said that the child had had an elevation of temperature to 101 F. the previous day. The stools were loose, and from three to four a day were passed. The turgor and muscle tone were poor. In performing a routine physical examination, a tympanitic note was obtained on percussion on the right side posteriorly, below the fourth rib. Auscultation over this area revealed distant amphoric breathing, with a metallic tinkle and splashing; otherwise, the lungs were normal. The liver was palpable one-half finger-breadth below the costal margin. The spleen was not palpable. The heart sounds were of good quality, and adventitious sounds were not heard. Cardiac dulness extended to 0.5 cm. outside the midclavicular line on the left and the midsternal line on the right.

In view of the negative physical conditions at the previous examinations, the possibility of an acquired diaphragmatic hernia on the right side was considered, and roentgen-ray study was advised.

Roentgen-ray study of both the chest and the abdomen in the dorsoventral position, after a barium meal, showed the heart and great vessels to be situated

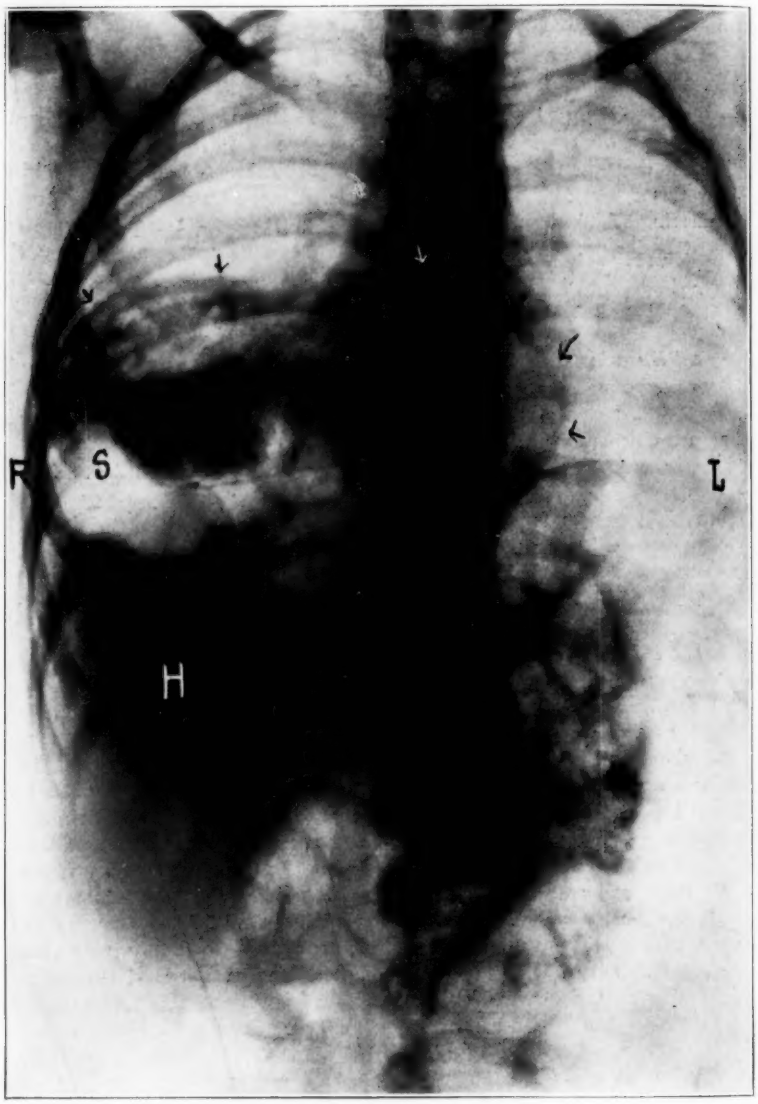


Fig. 1.—Ventrodorsal supine view; the arrows point to the right side of the diaphragm; *R*, indicates the right side; *L*, the left side; *H*, the liver, and *S*, the stomach, containing gas and barium.

normally. The left side of the diaphragm reached the fourth interspace anteriorly (ninth rib posteriorly). The stomach was noted above the liver on the right side and was capped by an arched line with a double contour. This line traversed the right side of the chest at a level corresponding to the second interspace anteriorly (sixth interspace posteriorly), extended downward slightly beyond

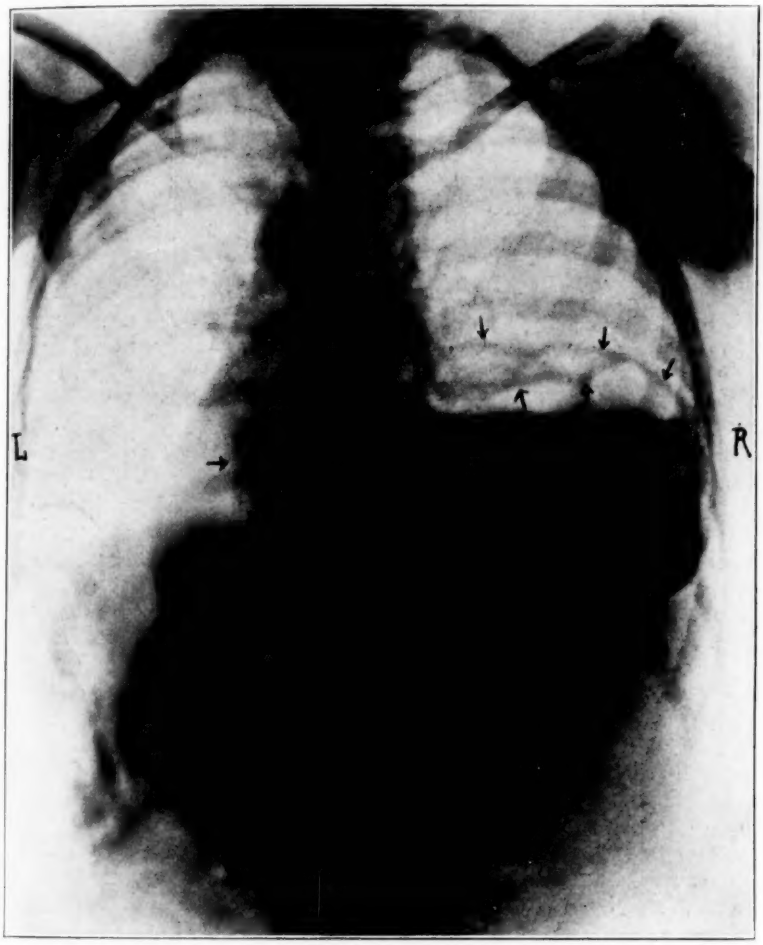


Fig. 2.—Dorsoventral erect view; the arrows point to double contour of the right side of the diaphragm.

the midline to the left side and descended to terminate at a point corresponding to the median insertion of the diaphragmatic leaflet on the left side (figs. 1 and 2).

A lateral view (figs. 3 and 4) showed the arched line as a single one without a break in its continuity; at its summit a steplike elevation was present. Its insertions, ventrally and dorsally, corresponded to the insertions of the diaphragmatic leaflet on the left side. Fluoroscopic observations revealed that the left side of the diaphragm moved normally. The arched right line moved synchronously

Fig. 3.—Right lateral recumbent view; the arrows point to the right side of the diaphragm; the arrow with the double head to the summit of the steplike elevation.

with the left, behaving in every way like a normal diaphragmatic leaflet, except that its motions were slightly diminished. The fact that the insertions of the arched line corresponded to the insertions of a normal diaphragmatic leaflet on the right side, and that its motions were those of a normal diaphragm, descending with inspiration and ascending with expiration, proved that it represented a relaxed but intact diaphragm. The liver was thought to be slightly lower in position than normally, its upper contour being slightly lower than that of the left leaflet. The colon, as outlined by gas, was normally situated. The mediastinum was not affected by respiration, no pendulum action being observed.

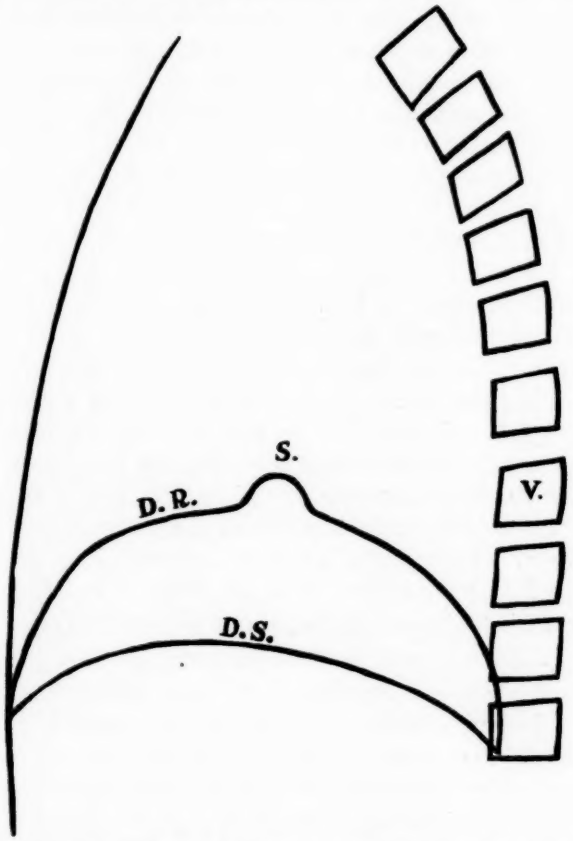


Fig. 4.—Schematic diagram for figure 3. *DR* indicates the right side of the diaphragm; *S*, the steplike elevation; *DS*, the left side of the diagram, and *V*, the vertebrae.

In view of the foregoing observations, a diagnosis of congenital nonrotation of the stomach with eventration of the right side of the diaphragm was made.

## COMMENT

Eventration is characterized by a general relaxation of one half of the diaphragm which allows the abdominal viscera to be displaced upward into the thoracic cavity. Although the diaphragm remains intact,

the stomach and intestines become lodged high up in the thoracic cavity and give rise to signs exactly like those observed in hernia. It has been shown at necropsy that in all instances in which eventration has occurred, whether on the right or the left side, the diaphragm is thin and translucent. A fringe of muscle fibers around the parietal attachments of the diaphragm is encountered in some cases. The affected side has an opaque, whitish color, in contrast to the red muscular aspect of the healthy side. There are about fifteen cases of eventration of the left side to one of the right. This is in contradistinction to hernia, in which the ratio is about four to one (Paillard,[6] Bowditch [10]). The theory that hepatic protection is the explanation of this is only partly true. The liver does not become a factor until after three months of fetal life. Hilaire [11] and Duguet [12] have demonstrated that the diaphragm in the fetus is not any more protected on the right side than on the left. This, according to Paillard, is only partly true, since the right side is definitely more protected after 3 months of age. One factor is certain, and that is that the right side of the diaphragm is formed first, and that it is definitely stronger than the left. This has been shown to be true by Bowditch [10] and Lacher.[13]

Kinney [5] explains the occurrence of the stomach on the right side in the following manner: "The stomach arises as a bulge from the esophagus at about 5 mm. development and remains in this position above the liver until after the fetus attains the 11-15 mm. stage. At this time the stomach rotates to the left and in the 17 mm. stage the two ends of the stomach have assumed the normal position, that is, below the liver." In our case, the stomach occupied the position of the organ in the 5-11 mm. embryo with the proximal end above the liver. It was impossible to determine the position of the distal end.

*Etiology.*—Most observers (Norris and Landis,[14] Bayne-Jones [1]) believe that eventration is invariably congenital. Some, however, believe that it may be acquired (Schlapper [15]). When acquired, it may be due (1) to injury of the phrenic nerve, as the result of infectious processes, pleural adhesions or tumors of the hilum, (2) to fatty or fibrous changes in the musculature of the diaphragm and (3) to differences in pressure on the two sides of the diaphragm. It is also believed that eventration

10. Bowditch: A Treatise of Diaphragmatic Hernia, Charleston Med. J. & Rev., 1855.

11. Hilaire: Les anomalies de l'organism, **1**:550, 1832-1836.

12. Duguet: De la hernie diaph. congénital, Thesè, Paris, 1866.

13. Lacher, quoted by Paillard (footnote 6).

14. Norris and Landis: Diseases of Chest, Philadelphia, Saunders & Company, 1920, p. 638.

15. Schlapper: Zur differential diagnose der Relaxation und Hernia Diaphragmatica, Beitr. z. Klin. d. Tuberk. **66**:260, 1927.

of the diaphragm may occur after prolonged and sustained increased intra-abdominal pressure. The fact that eventration occurs more frequently on the left side, i. e., the weaker side, that it is encountered in the fetus and in the new-born, that it is associated with other congenital anomalies and that the lung on the affected side is not compressed, all point to its congenital origin. In our case, the absence of symptoms over a long period of time, the occurrence of the stomach on the right side above the liver, the absence of signs of compressed lung and the comparative youth of the patient, all point to the congenital origin of this condition. A severe attack of whooping cough with marked and frequent paroxysms, such as our patient had, might produce a simple eventration as a result either of increased intra-abdominal pressure or of dysfunction of the phrenic nerve, or of both. In whooping cough, injury to the phrenic nerve may result from the concomitant enlargement of the tracheobronchial nodes. Neither one of these factors, however, could possibly explain the abnormal position of the stomach.

*Symptoms.*—Vomiting and developmental retardation are practically the only constant symptoms encountered. Kinney [5] and Eppinger [3] observed them in their cases. Harris and Stivelman,[7] however, did not mention vomiting, but observed marked emaciation in their patient. Complaint of pain in the upper part of the abdomen is also encountered, but this is not a constant symptom. Kinney's patient, in addition to the vomiting, also had marked dysphagia. Constipation may or may not be present. On the other hand, symptoms may be entirely absent. In fact, Norris and Landis [14] stated that the condition is usually discovered accidentally during a routine physical or roentgen-ray examination or at autopsy. It is their belief that the condition is always congenital and that symptoms are not produced because the development of the thoracic and abdominal organs compensate for the abnormality. In our case, anorexia, vomiting and failure to gain in weight were the only complaints.

*Physical Signs.*—Physical signs depend on whether the liver or the stomach has ascended into the thoracic cavity. If the liver is high up, there is flatness of the right side posteriorly with absence of vocal fremitus and breath sounds, from the third or fourth rib downward. It is evident that, under such conditions, a mistaken diagnosis of thickened pleura or fluid in the pleural cavity may easily be made. In cases of eventration with nonrotation of the stomach there is normal breathing and a normal percussion note down to the third or fourth rib anteriorly and to the sixth or seventh rib posteriorly; below this point, percussion gives a tympanitic note. Vocal fremitus is absent, but whispered pectoriloquy and distant amphoric breathing with splashing and metallic sounds are usually heard. If the stomach is full, however, the tympanitic note may be replaced by flatness, which is shifting in

character. In other words, the physical signs change with the administration of food and with the position of the patient. The heart is not displaced, as generally occurs in hernia and in hydropneumothorax. Evidence of compression of the lung is also absent, as a rule. These two factors account for the absence of cyanosis and clubbing of the fingers—conditions found when hernia is present.

*Diagnosis.*—Obviously, from a therapeutic point of view, it is essential to differentiate eventration of the diaphragm from hernia of the diaphragm. Unfortunately, roentgenologic examination alone is not always a deciding factor. At times fluoroscopic observation of the movements of the diaphragm will help. In eventration, normal movement with inspiration or reduced movement, depending on how much muscle is normal, is usually observed. In hernia, paradoxic movements of the diaphragm occur, due to the fact that the negative pressure is increased in the chest during inspiration, which sucks up the hernial sac. If there is complete degeneration of the phrenic nerve, however, paradoxic movements will be encountered in eventration; also, if the hernial sac is adherent to the diaphragm, the movements of the diaphragm will be normal. In eventration, there is increased intragastric pressure during inspiration, as occurs under normal conditions. In hernia, the opposite is true, i. e., the intragastric pressure decreases with inspiration and increases with expiration. In eventration, the diaphragm, although high up, shows a smooth shadow arching upward across the thorax, and it has a normal contour; in hernia, irregularities and bulging of the diaphragm may be observed. Double contour of the diaphragm has been observed in eventration. It has been commonly accepted that the upper line is the diaphragm and the lower line, the greater curvature of the stomach. Assman [16] stated, however, that the existence of a space between the diaphragm and the stomach is impossible, and it is his opinion that the double contour is due to a steplike formation of various portions of the diaphragm. A double contour is noted in our case in the anteroposterior view (fig. 1). The lateral view (fig. 3) demonstrates clearly how this double contour is produced. In this view, a steplike elevation at the dome of the diaphragm is seen. The upper line of the double contour in the dorsoventral view represents the peak of the elevation seen in figure 2, the lower line representing the remainder of the dome. Thus actually no space exists between the two lines. This elevation may be produced by either one of two factors: (1) normal but irregular muscular contractions or (2) balooning of the weakest portion of the diaphragm. The absence of double contour does not necessarily rule out eventration. Götze [17] has advised the employment

16. Assman: Die Roentgendiagnostik der inneren Erkrankungen, p. 291.
17. Götze, quoted by Schlapper (footnote 15).

of pneumoperitoneum to differentiate the two conditions. This procedure, however, is not without danger, and is valueless if there are adhesions between the sac and the diaphragm. Funk [18] found an exaggerated inspiratory flaring of the costal margin in eventration on the affected side; in hernia, on the other hand, there is inspiratory fixation of the left costal margin of the subcostal angle. His explanation of these observations is that the high position of the diaphragm in eventration places it at a mechanical disadvantage and therefore permits greater flaring than on the opposite normal side. Funk was able to differentiate two cases by these observations. For differentiation, Beltz [19] employs the following procedure: Air is introduced into the stomach by means of a catheter or stomach tube, and the contour of the stomach is observed fluoroscopically; in hernia the stomach, when it becomes distended, approaches the thoracic wall. The air is then aspirated while the patient is still behind the screen, and it is observed that the stomach moves away from the thoracic wall. The stomach also assumes various contours with the introduction and aspiration of air and with changes in the position of the patient. In eventration, however, a regular arch immediately over the stomach "air bubble" is always observed. In hernia, as a rule, the heart is displaced.

Finally, it must be stated that the differential diagnosis cannot always be made with certainty, even when all the foregoing procedures are followed. Latze [20] carefully observed a case of supposed eventration for several years, which at autopsy proved to be a hernia.

In our case, a diagnosis of eventration with nonrotation of the stomach, rather than hernia, is logical for the following reasons: (1) the abnormal position of the stomach on the right side, above the liver; (2) the presence of a relaxed but intact diaphragm above the stomach; (3) the normal, although slightly diminished excursions of the diaphragm; (4) the presence of double contour in the dorsoventral view; (5) the absence of mediastinal displacement; (6) the absence of compressed lung, and (7) the clinical course. At no time were severe symptoms or signs present, as cyanosis, clubbing of the fingers, dyspnea and other symptoms pointing to respiratory and cardiac embarrassment.

*Differential Diagnosis.*—This condition must be differentiated from hydropneumothorax, subphrenic abscess with accumulation of gas and cavitation of the lower lobe of the right lung. Eventration of the right side has been most often mistaken for hydropneumothorax and subphrenic abscess. Harris and Stivelman [7] considered their case one of

18. Funk: Congenital Diaphragmatic Hernia, M. Clin. N. A. **8**:749, 1924.

19. Beltz: Differentialdiagnose zwischen Hernia and Eventratio Diaphragmatica, München. med. Wchnschr., 1910, p. 1006.

20. Latze: Ueber Eventratio Diaphragmatica, Deutsche med. Wchnschr. **32**: 1622, 1906.

hydropneumothorax for four months, when a barium meal revealed the true nature of the disturbance.

In Dietelin and Knierim's [21] patient (hernia of right side of diaphragm with herniation of stomach), a diagnosis of subphrenic abscess, resulting from a perforated gastric ulcer, was made, and exploratory puncture was performed twice. Each time a cloudy yellow fluid containing many leukocytes was aspirated; this was undoubtedly gastric juice. Before resorting to diagnostic puncture or aspiration, eventration of the diaphragm with nonrotation of the stomach should be ruled out, particularly in the absence of constitutional symptoms and of pathologic processes which may give rise to either subphrenic abscess or hydropneumothorax.

*Prognosis.*—Jones [1] stated that after the first year eventration is rarely a cause of death. It is compatible with life, cases having been reported in persons between the ages of 20 and 50 years (the cases of Harris and Stivelman, Eppinger, and Jones). The danger of incarceration of the displaced abdominal viscera, however, is a factor to be kept in mind.

## SUMMARY

1. A case of eventration of the right side of the diaphragm with nonrotation of the stomach, congenital in origin, is reported.

2. The differential diagnosis between eventration and hernia of the diaphragm is discussed.

3. The possibility and danger of confusing eventration with nonrotation of the stomach, with spontaneous hydropneumothorax and with pyopneumothorax subphrenicus are pointed out.

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21. Dietelin and Knierim: Hernia diaphragmatica dextra, Berl. klin. Wchnschr., no. 25, p. 1174, 1910.

# Progress in Pediatrics

## EPILEPSY IN CHILDREN

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Just what epilepsy is, no one knows. That it is polymorphous, is notorious; that it is still frequently unrecognized and, consequently, that patients are maltreated, is a fact and a reproach to the medical profession. In this article I have restricted myself to abbreviated records of some instructive examples of this strange malady in children,[1] and a few comments on the treatment. In addition, I wish to protest most emphatically against the too prevalent light and casual consideration that is given to such little "spells" as I shall describe. Such indisposition merits the same grave attention as do pathogenic germs, which also are small but potent for harm.

### REPORT OF CASES

CASE 1.—A boy, aged 11, began to have momentary dizzy spells, without falling, from once a day to once a month when he was 4 years old. At 6 years of age the attacks became more severe; dizziness was followed by a few minutes of confusion. They have occurred once a day for the past few months.

CASE 2.—A boy, aged 13, began to have momentary dizzy spells at 8 years of age; he would say "Papa, I'm dizzy," and at once, "I'm all right now." A whole year intervened between the first and second attack. Gradually the attacks became more frequent, occurring two or three times a day. At 8 years of age, he began to fall behind in his school work. During the last year he has also had nocturnal attacks.

CASE 3.—A man, aged 25, had had attacks since childhood in which he seemed dazed for a moment and everything appeared blue. These attacks occurred only once in from three to eight weeks. No grand mal[2] attacks occurred till he was 21 years of age.

CASE 4.—A man, aged 24, had had slight attacks without loss of consciousness in which "all around him would become dense," from the age of 12 to the age of 17; he would be slightly dizzy and would breathe heavily. These attacks occurred only about once in four months. After the seventeenth year, they became more severe, although they were never associated with falling. The first major attack occurred when the patient was 21.

CASE 5.—A man, aged 43, had had slight dizzy spells nearly every morning since the age of 13. The dizziness was preceded by a transient dead, dull ache in the sacral region. These attacks were "so slight he paid no attention to them."

1. These case reports are abstracted from a paper by Dr. David M. Levy and myself which will be published later in the *Archives of Neurology and Psychiatry*.

2. By grand mal, or major attacks, I mean a general convulsion with complete loss of consciousness.

Three months before, after a hot day, they became distinctly worse and were followed by light-headedness and sometimes by staggering.

CASE 6.—A girl, aged 14, had had a number of fainting spells during childhood; at 13 years of age, the first grand mal attack occurred.

CASE 7.—A young woman, aged 21, had had slight dizzy spells several times a day from the time she was 14 until she was 19. Two to three times she fell, but arose at once. There was no loss of consciousness. A typical grand mal attack began when she was 19.

CASE 8.—A girl, aged 16, sixteen months before examination noticed a sudden blackness while combing her hair; she dropped the comb and was dazed for a few seconds. During the next fourteen months, she had five more of these "dazed spells," then grand mal attacks occurred.

CASE 9.—A youth, aged 18, six months before examination awoke suddenly at night frightened. On a hot day, three weeks later, when very tired, he had a sudden feeling of faintness and an orange which he had been holding fell out of his hand. During the next six months, he had had ten such attacks. Two months after the beginning of these attacks, he had a distinct petit mal attack, and two months after that, grand mal seizures.

CASE 10.—A woman, aged 32, had had scarlet fever with nephritis at 6 years of age; during this illness she had had three convulsions. Soon thereafter she began to have momentary attacks of pressure sensation in the epigastrium with nausea but she did not vomit. These attacks recurred every two or three days until she was 11, when menstruation began. Before the first menstruation, she had had severe hemicrania with vomiting, and it was said that she "probably was unconscious for a time." Similar headaches had continued to recur about one week before each menstrual period, until the age of 17, when typical petit mal attacks began, the aura for every seizure being a sensation of epigastric pressure.

CASE 11.—A young woman, aged 19, at the age of 8 began to have attacks of gas in the left iliac region with excessive flow of saliva, followed by belching; these attacks lasted "about ten minutes" and recurred about once a week for about three weeks. She did not have an attack for a year, when they began again, and she continued to have about one a week until she was 14 (six years), when grand mal attacks began.

CASE 12.—A man, aged 42, began to have slight attacks at the age of 8 which started as a feeling of nausea in the chest. The sensation would rise to the head and be followed by a few moments of confusion in which he would try to talk, but could not express himself well. There was never loss of consciousness. These attacks recurred about every two weeks to two months until he was 42, that is to say, for thirty-four years, when he had a fully developed grand mal attack.

CASE 13.—A young woman, aged 24, at about 12 years of age began to have momentary attacks of what she called "nausea" starting in the epigastrium and going upward. There was no loss of consciousness, dizziness or confusion. These attacks occurred about once a month, but gradually increased until, at the age of 20, she was having two or three a month. At 22, she suffered the first complete attack, which started with this sensation as an aura.

CASE 14.—A man, aged 36, at about 14 years had had half a dozen brief attacks of "queer feelings" in the abdomen, followed by headache. Some years later, he began to have petit mal attacks in the form of short mental lapses in which he "did not lose consciousness" but was confused, stopped whatever he

was doing and "was afraid to speak." These have continued, and a year ago he had his first grand mal attack.

CASE 15.—A girl, aged 9, since the age of 7 has suddenly turned pale about once a month. For the last few months, the attacks have been more pronounced and have been followed by sleep. Six months following the onset, she had the first severe seizure in which she fell and had involuntary evacuation of the bladder.

CASE 16.—A man, aged 24, had had occasional attacks of momentary pallor with slight confusion when he was 10 or 11 years of age. These attacks were noticed several times within a year. Then apparently he was well until the age of 18, when he developed petit mal attacks, and at 24 he had complete epileptic seizures.

CASE 17.—A man, aged 30, had had several brief attacks in which the tongue, lips and throat seemed to be paralyzed; he could make only inarticulate sounds and drooled a little. These attacks occurred between the age of 10 or 11 and 13 or 14, i. e., for about three years. The attacks lasted only a few seconds. He was known to have had one at night. At 13, the first grand mal attack supervened.

CASE 18.—A man, aged 24, since the age of 14 had had attacks of a creeping sensation about the right corner of the mouth lasting a few seconds. These attacks occurred only from once in two weeks to once in six months and were not accompanied by any other sensation or symptom. For two years, from the age of 19 to 21, he had not had any attacks. Then they recurred and increased in frequency until they occurred daily; at 24, he had his first general convulsion starting at the right corner of the mouth.

CASE 19.—A girl, aged 10, began to have peculiar spells at the age of 8, in which she would suddenly run to the mother and say "water." If in school she would run to drink water. She felt dizzy during these attacks. As soon as she swallowed the water, she would feel all right. The first major attack occurred at 10 and began with an aura of thirst.

CASE 20.—A girl, aged 13, had had "cross spells" since the age of 2, which came on suddenly without ascertainable cause and lasted from an hour to several days. At 6 or 7 years of age, she would scream in her sleep about every three months, and her mother would find her limp. Typical major attacks developed at 8 years, and when she was 9 petit mal attacks occurred.

CASE 21.—A man, aged 30, began to have sudden screaming spells at the age of 2, in which he would lie on the floor, kick and dig at his arms. These lasted from thirty seconds to a minute; then he would run off and play. The attacks ceased at the age of 5. From the age of 1 year and through childhood the mother had noticed that once or twice a week the child would roll his eyes for a second or two. Minor attacks began at 14 and major ones at 20.

CASE 22.—A youth, aged 19, had begun to have momentary attacks at the age of 3 in which he staggered and laughed; apparently he was always conscious. The attacks occurred several times a week, but diminished in frequency until they were seen only several times a year when he was 15 or 16. Major seizures began when he was 18.

CASE 23.—A girl, aged 13, had begun to have unprovoked spells of laughing and crying at 11 years of age. At the same time she developed sleep-walking; with this swallowing movements were sometimes noticed, and she would fuss with the bed clothes; she also began to wet the bed two or three times a week. Since the age of 11 she had had headaches with occasional nosebleed. Major seizures began at 13.

CASE 24.—The patient was a boy, aged 6, who at 5 years of age, while at play suddenly ran to his mother and said "Who's shot?" though no sound had been heard. He seemed frightened for a few minutes, and then was all right. Five or six days later, he ran to the mother as before, was unable to talk and "seemed oblivious to surroundings." In a few moments, he was normal. During the following few months, similar attacks appeared, and then major seizures began.

CASE 25.—A youth, aged 21, had begun to have sudden attacks of "lonesome feelings" at 9 or 10 years of age, which lasted only from ten to fifteen seconds, without loss of consciousness. During these attacks all objects appeared blue. At 11 years of age, these attacks began to terminate in general convulsions with loss of consciousness.

CASE 26.—A woman, aged 42, had begun to have attacks of sudden "homesick feelings" with an indescribable dreamy state, discomfort around the lower thorax and a "very sorry" feeling when she was 5 or 6 years of age. These attacks continued to recur. They were of only momentary duration and occurred once a month to several weekly. There had been no loss of consciousness until a month before this article was written, when these "little fits" began to eventuate in general convulsions.

CASE 27.—A woman, aged 48, since childhood had had attacks of a dreamy state about once in 6 months in which she saw beautiful scenes or past acquaintances. Four years ago, she began to have minor and major attacks of epilepsy.

CASE 28.—A man, aged 58, had transient "frightened feelings" during childhood. At 51, he began to have attacks at night of a strange feeling about the nose and throat and a peculiar taste "like the odor of a cat's nose." At first this was rather pleasurable, then it became disagreeable. About two years ago, the first nocturnal convulsion appeared.

CASE 29.—A man, aged 23, had had scarlet fever when a child and soon after, at 7 years of age, he began to have transient attacks of "queer feelings" without loss of consciousness. They occurred two or three times a month, with free periods of from six to twelve months until the onset of minor and major attacks at 17 years of age.

CASE 30.—A boy, aged 16, since the age of 11, had had attacks of a "feeling of apprehension" every other day or so lasting a few seconds. At 13 he began to have attacks of rigidity with a fixed stare; the fingers of the right hand would drum, although he said that he could control this. His head and eyes would turn to the left, and in more severe attacks he would stagger in circles. He thought that he did not lose consciousness in these attacks, which occurred every four or five days.

CASE 31.—A woman, aged 28, had begun to have attacks of "lonesome feelings" several times a day at 16 years of age; these attacks were associated with precordial oppression and palpitation. They continued to occur until she was about 18. Major seizures began when she was 22, and the minor episodes also returned.

CASE 32.—A girl, aged 14, as a small child had gone to her mother, saying "I'm sick;" she would lie down for a few minutes and get up apparently well. At 8 or 9 years of age, while standing near a table, she said she "felt bad," dropped to her knees and promptly got up. The whole episode lasted only a few seconds, and apparently there was no loss of consciousness. Such attacks recurred three or four times daily for some time, and then disappeared entirely for six months. When they recurred, they were more severe and were attended by loss of consciousness.

CASE 33.—A boy, aged 3, when 2 years old had suddenly awakened "shivering as though with cold." This lasted possibly half an hour, probably much less. A week later, he had a similar attack. A few months later, major convulsions appeared, and these have continued.

CASE 34.—In this case, the patient was a girl, aged 16. At 9 years of age, while walking with her mother, she said she had to stop for a moment. She did so, and immediately was normal. Such peculiar episodes were noticed every two or three weeks. After about a year, the mother noticed that during this act the mouth was drawn to one side, and soon thereafter the attacks began to eventuate in a fall and unconsciousness.

A word to the wise is sufficient. After submitting this list of thirty-four cases, I need not elaborate my thesis of accurate and detailed anamnesis, careful examination, prompt and vigorous treatment.

What do I mean by vigorous treatment? The report of a case, quoted from another paper by Dr. Levy and myself, will serve as an example.

At 3½ years a rather frail, constipated boy, whose digestion was poor and who easily took on infections, began to have slight attacks. One of our greatest internists cared for his general condition and gastro-intestinal tract wisely and faithfully, but seemed to be amblyopic for the fits. After a few months the boy was worse. It took almost a year to convince the parents that the fits had to be treated—even though he did frequently vomit after one and often had offensive stools. On full and continuous doses of bromides and hygienic treatment he improved at once, made a good recovery, and has remained well for twenty-one years.

The diet, exercise, bowels, sleep and emotional life should receive adequate attention. Ordinarily this means a great deal of work with the parents, seeing the child frequently and patient, detailed supervision. In short, the physician must remain on the job, which, in my experience, he rarely does.

The matter of medication is ultrasimple in principle, but sometimes ultradifficult in execution. The treatment for epilepsy is empiric. The object of medicine is to suppress the fits and break up the habit, if the patient can take care of a sufficient dosage to permit its accomplishment. I rely almost entirely on sodium bromide and phenobarbital. I think that bromide is better for children than phenobarbital. Many physicians seem to avoid the use of bromide. Its use may be and has been abused, but to be afraid of it in full doses is foolish. Indeed, not infrequently I find it a good plan to give the child bromide to start with until the fits are stopped and then reduce the dose. Sometimes a combination of bromide and phenobarbital is more effective than either alone: bromide twice a day and phenobarbital once a day. For children with cold hands and feet, especially if the children are of the heavy, sluggish type, I like to add one dose of thyroid extract daily.

I believe, though I could not prove it, that many epileptic children and young people do not normally digest proteins well. Possibly this is part of their rather frequent tendency to overeat. They are prone to have indican in the urine; consequently, they need a great deal of exercise, reduction of the amount of meat in their diet and constant supervision of their intestinal condition.

Briefly, the successful management of a case of epilepsy means a prolonged, vigorous campaign without intermission; one must be on duty all of the time. Even skilful and experienced pediatricians have been known to take the convulsions of children lightly; they just attend to the general health or grow careless as to medication and control when there has been no recurrence for a year or so.

I have no sympathy with the theory that the cause of epilepsy lies in emotional conflict, and with the consequent proposal that epileptic persons should be psychoanalyzed. Nor have I been able to confirm the often repeated assertion that there is such a thing as the epileptic temperament or character. This attitude is, of course, not inconsistent with the obvious fact that no person can have epilepsy without a certain underlying cellular instability, the nature of which is unknown.

# Pediatric Biographies

## CORNELIUS ROELANS
### 1450-1525

JOHN RUHRÄH, M.D.
BALTIMORE

Roelans (or Roelants), who wrote the text of the third incunabulum, was a Flemish physician, born in Mechlen in 1450. At the age of 16 he entered the University of Löwen, and while there, or on subsequent visits, he must have made good use of his time in studying the books and manuscripts of the early writers. Later, he returned to Mechlin, where he practiced until he died, in 1525. In 1494, he married Caecilie von Duffel, who bore two children. He must have been a physician of ability as well as one of learning, for he was "Hospital und Stadtarzt."

His book is a rare one, but fortunately it is now available through the efforts of Karl Sudhoff, who, in his "Erstlinge der Pädiatrische Literatur," has reproduced the text in facsimile and accompanied it by an illuminating essay. The original may be seen in the library of the University of Leipzig, and there is also a copy in the Hunterian Museum at Glasgow and a few leaves at Cambridge, England.

The work of Roelans, though preserved intact, was evidently printed and bound with some other book, the nature of which is not known. It is a folio of small size, consisting of 117 printed pages. It was evidently printed by J. Veldener of Löwen (Louvain), and is dedicated to Philip I, the Handsome, of Burgundy, Brabant and Flanders. The author's name appears on the second page.

In the course of the work, he refers more than once to "the little book on diseases of children." Sudhoff found, in various libraries, fragments of this manuscript work entitled, "Incipit practica passiones puerorum adhuc in cunabulis iacentium" (Diseases of Children Still in the Cradle). This work is a different manuscript from the "Liber de passionibus puerorum Galeni," which Sudhoff believes to be a spurious work, not written by Galen, but compiled much later. Both of the manuscripts had a wide vogue during the middle ages and doubtless exerted a considerable influence on pediatric thought and practice, as is evidenced in the case of the first one by the high esteem given it by Roelans centuries after it was written. In addition to "the little book," Roelans quotes a long array of ancient and medieval writers. Indeed, he seems to have gone through the medical literature thoroughly, picking out the pediatric items and bringing them together in a single

# Ecciii

vi nature transmissa ¶ Causa est superfluitas nutrimẽti in quãto seu quali sumpta ex qua superfluitas aquosa grossa vel calore debili ventositas generatur que ad cutim transmissa generat in toto vel in aliqua parte corporis inflationem ¶ Signa patent ¶ Cura ¶ Regimen nutrici temperatum inungatur tunc infans cienur remedio quod in quodam libello egritudinum puerorum inueni Recipe summitates sambuci et ebuli coque in vino albo et pannis calidis puerum inuolue secundum totũ vel secundum partem epithimando sic suple eũ

¶ Quinquagesima secunda egritudo puerorum et vltima est nimea maciencia seu extenuatio puerorum

¶ In quodam libello egritudinũ puerorũ

Si vero puer fit nimis macilentus habeat ꝙ tantum ossa et pellem et multũ fit debilitatus ¶ Cura est fiat ei tale balneum Re caput arietis et pedes coquantur in aqua dulci sic ꝙ carnes separẽtur ab ossibus et in tali aqua infantis balneetur et cum exierit a balneo tali vngento vnguatur Recipe butiri olei rosati vel violati vel communis aua ʒ xx et de pinguedine cruda de carnibus porcinis ʒ x cere albe ʒ viij qualibet die post exitum balnei vngua tur totũ corpus hoc vnguento et multũ inpinguat

¶ Et sic est finis capituli de regimine infantis

A page from Roelans' book. (Courtesy of Professor Sudhoff.)

volume. The authorities most quoted are Hippocrates and Galen, but mention is made of Dioscorides, Rufus and Sextus Placidus; of the Arabians, Avicenna, Rhazes, Mesue, Serapion, Honein, Avenzoar, Hali Abbas and a great number of medieval commentators, Johannes Matthews de Gradi, Nicolaus Florentinus, Solanus, Jacobus of Forli, Gentile de Fulgineo, Franz von Piemont, Marsilius de Sancta Sophia and Jacques Despars. The range is wide and mention is made of Gerardus Cremonensis, the "Conciliator" of Peter of Abano, the "Lilium" of Bernhard von Gordon, the "Regimen Sanitatis," John of Gadesden's "Rosa Medicinae," Gilbertus Anglicus, Arnold of Villanova and others. Roelans laid great stress on the opinions of Avicenna as expounded by Jacques Despars, a Paris physician, who wrote a long treatise on that Arabian worthy, and who divided the original text into chapters.

Roelans lived in an age of authority, and he accepted what was written, but he was neither the first nor the last medical fundamentalist. His compilation is a valuable one, as it gives a fair picture of the pediatric thought and practice of his time and, through his references, to the period that preceded him. One of the chief lessons to be learned from Roelans is the sterility of static thought. As long as the authorities are accepted and followed blindly, advances cannot be made. From time to time, periods for rest and reflection are needed to digest the accumulated new material, to assimilate what is good and reject what is useless; but growth, that is progress, can only come by adding new material, and this the age of Roelans failed to do.

Roelans' work was reissued by Sebastian Ostereicher, who will be considered later.

Some idea of Roelans' style may be gleaned from the following excerpt taken from the English translation made for me by Dr. Herbert F. Wright, about half of which will be found in "Pediatrics of the Past".

"Avicenna also offers a medicine extremely beneficial in wind of infants, such as give in a potion to the infant a weight of three grains of barley of powder made from dry origan and castoreum and cumin mixed equally and finely ground. Jacques Despars adds: 'And it may be given with milk or with water of honey. For the said powder is strongly carminative and resolves ventosities whether they have been in the stomach or in the head or in the intestines.'

"Note here the doubt of Jacques Despars to the following effect. But it is worthy of admiration that the eminent Avicenna here in wind of infants employs medicines of vigorous warmth, as castoreum, cumin and origan, and in atas of infants recommends cool medicines both inside and outside, as water of barley and its savich and spodium and seed of portulacca and also violet placed upon the head, which in no wise seem to be competent in inflation. Nor does Rases, the highest and greatest practitioner in the cure of diseases, recommend cool medicines in this case but many warm ones, as oil of bitter almonds, oil of ben and water

of marjoram gently introduced in the infant's nostrils and a plaster of oliban, sarcocolla, serapinum of gum of almonds placed upon the head and ointment of nucum and smeared upon the head with a little oliban and wolf's gall and a little amber of myrrh introduced in the nostrils, just as is maintained in the fifth species of diseases of children.

"For answer, note that ventosity is twofold. One is vaporable from the weak heat ventilating the humidity. The other is smoky, turbid, which a vigorous heat generates operating against the humidity. And in the extermination of the former, warm medicines are beneficial, and in the extermination of the latter, cool medicines."

# News and Comment

## JOURNAL IN ENGLISH FROM VIENNA

We are in receipt of the journal *Ars Medici,* which is published by the American Medical Association of Vienna.

It is the only journal published in English on the continent of Europe at the present time. The subject material consists of review articles and abstracts from the various branches of medicine, and each number contains some pediatric reviews. The journal is published monthly and the cost is $3.00 a year. The price of the first five volumes together with the subscription for 1928 amounts to $15.00. For one who wishes to get special information in the various branches of medicine, the journal is well worth while.

## NEW DEPARTMENT AND GIFTS AT STANFORD UNIVERSITY

Pediatrics, which previously had been a division of the department of internal medicine, has been made a full department in the medical school at Stanford University. The school has received $500.00 from Mr. Edward M. Mills for free beds for children and $610.69 from the estate of Miss Ann Whitney Sperry to be used for the benefit of Protestant and Catholic crippled or orphaned children of San Francisco.

## FUND FOR STUDY OF INFANTILE PARALYSIS

Announcement is made that $5,000 annually has been given to the University of California to promote investigation of infantile paralysis or other diseases of children.

## APPOINTMENT OF DR. BRET RATNER

Dr. Bret Ratner has been appointed clinical professor of pediatrics and lecturer in immunology at New York University and Bellevue Hospital Medical College.

## DEATH OF PROFESSOR GURGEL

On Jan. 2, 1928, the eminent pediatrician, Prof. Luis Nascimento Gurgel, died in Rio de Janeiro.

# Abstracts from Current Literature

## Anatomy and Physiology

THE PHYSIOLOGY OF BONE GROWTH. HUGO MAAS, Arch. f. klin. Chir. **146**:786, 1927.

The increase in thickness of bone depends on the periosteum, the length on the growing cartilage. In order to understand the physiologic significance of growing cartilage one must know that the growth in length is somewhat different from the growth in thickness. Length does not occur in the same fashion as thickness when it results from the deposition of layer on layer, but results from the formation of a spongelike network, which constitutes a far more important rôle against mechanical injury than the compact cortex. The formation of the spongiosa layer is laid down as soft calcium-free osteoid tissue, which after mechanical activity becomes the seat of deposits of calcium salt, and by virtue of the deposition of calcium there is exerted a physiologic growth pressure. If it were not for such a factor, which seems to possess a protective faculty, the spongiosa would even stop growing because of the mechanical activity. The whole question is one of growth of bone taking place most in that part of the bone which sustains the greatest amount of function; this is equally applicable to both cortex and spongiosa.

Formation of cartilage at the growing ends of bone is in proportion to the rate of ossification, and the bone is laid down in the direction of the force. Therefore, the length of bone is dependent directly on the intensity of the growth of cartilage. This growth in length is most marked between the ages of 3 and 5 years, and again between 14 and 17, at which periods the epiphyseal line is widest, for the rate of thickening cannot keep pace with the rate of lengthening.

The same is true in pathologic conditions in which there are mechanical hindrances in the ossification process of the growing end and, as a result, widening of the epiphyseal line occurs. Any factors interfering with normal growth of bone, such as trauma or disease (prenatural ossification, chondrodystrophy, chondromatosis, cartilaginous exostoses), will result in a growth that is asymmetrical.

The author discusses an "expansile progression" of growth of bone. On this, the growing pressure exerts a physiologic influence, in which fundamental processes are at work that with each bodily activity exert a certain significance

OBERHELMAN, Chicago.

IS THERE A FEVER DUE TO EATING, THIRST AND CRYING? HANS RIETSCHEL, München. med. Wchnschr. **73**:2057 (Dec. 3) 1926.

Rietschel distinguishes between a rise of temperature due to central influence and so-called hyperthermia. He considers the former "real" fever. The latter is ascribed to the lack of sufficient water for regulation of temperature and can be brought about by diminished intake of water and increased intake of protein. It yields quickly to administration of fluid. Bodily exercise, which is known to increase the metabolic rate, may, under conditions of ordinary thirst, lead to a greater increase in temperature. Rietschel concludes that there is a fever due to eating, thirst and crying but that these are not real conditions of fever but are really hyperthermia due to physical dynamic stasis of heat. The condition which leads to this stasis may be combined at times with real fever and may increase it.

AMBERG, Rochester, Minn.

## Pathology, Bacteriology and Biochemistry

CENTRAL FACTORS IN HUNGER. FREDERICK HOELZEL, Am. J. Physiol. **82**:665 (Nov.) 1927.

A full discussion relating to the theory of hunger, based on certain personal experiences of the author, and a careful survey of the literature is presented in this paper. The conclusion is reached that, fundamentally, hunger is centrally rather than peripherally or gastrically determined. This belief is based first on the observation that when the desire to eat is suppressed by excessive food ingestion, the following gastric (hunger) contractions are noted as local (epigastric) feelings unattended by "hungriness," also the fact that hunger is noted to occur after fasting independently of gastric movement. The common reference of hunger to the stomach is dependent more on the epigastric seat of the so-termed incidental hunger factors, as gastroduodenal acidity and gastric emptiness in relation to periodic contraction of the stomach, rather than on such locale for the specific phenomenon or urge of hunger as such. This conception of an essentially central source or origin for hunger is interesting and renders more easily understood such observations as the lack of hunger in certain animals during sexual heat (in seals and salmon), the persistence of eating when the stomach has been removed, and the commonly observed hunger-inhibiting effect of fever, affective activity and psychic preoccupation.

RAPHAEL, Detroit.

THE RED CELL SEDIMENTATION TEST IN CHILDREN. E. M. GREISHEIMER, J. A. MYERS, N. P. PETERSON, A. D. KLEIN and A. COLLINS, Am. Rev. Tuberc. **16**:344 (Sept.) 1927.

Red cell sedimentation tests were made on a group of 152 children whose ages ranged from 6 to 17 years, and who were all attending school regularly. In practically every case, the roentgen ray showed moderately enlarged calcified lymph nodes at the hilum, and masked juvenile tuberculosis was the usual diagnosis.

Correlation was not found between the age and the index of red cell sedimentation. The latter seems higher in children than in adults.

Marked differences were not found between boys and girls.

Few children showed a positive Larson ring test, but these showed a faster sedimentation than those having negative tests.

The boys with positive Pirquet or Mantoux tests showed a faster sedimentation rate than those having negative tests, but the condition was reversed in the girls.

SMITH, Ogden, Utah.

THE METABOLISM FOLLOWING INJECTION OF DIPHTHERIA TOXIN IN DOGS. HUGH W. JOSEPHS, Bull. Johns Hopkins Hosp. **41**:400, 1927.

In 1909, Howland found evidences of destruction of body proteins in patients with diphtheria.

The present study was made on dogs following the intravenous injection of diphtheria toxin.

The author's summary follows:

"As one sums up what has been accomplished by this study, three facts stand out. The first is the demonstration of toxic destruction of protein in an afebrile period. Whether this destruction of body protein contributed to the severe symptoms suffered by the dogs, I cannot say from the results of these experiments. One may conjecture that the heart muscle has furnished its quota of the increased nitrogen excretion, but direct evidence of this is lacking, for weakness of the heart muscle was not a feature in the course of the intoxication, as it was in the case of children studied by Dr. Howland. It is probable that the increased urinary nitrogen comes largely from muscle tissue, and the lowering of resting metabolism during the intoxication period

may merely be further evidence of this. The second fact is the prevention of the increased nitrogen excretion by a diet high in carbohydrate. The mechanism of this action is not clear. If carbohydrate prevents the destruction of muscle protein, why does it not then diminish the excretion of creatin as it does when given during starvation? This effect of carbohydrate is deserving of further study, not only the effect on nitrogen excretion but also the effect on the actual pathological process and the course of the intoxication. The third fact is the demonstration of a period of latency, the duration of which bears an inverse ratio to the severity of the intoxication. Fourthly, the tendency to low temperature was studied and is ascribed to two influences. One is the vasodilator action of the toxin as a result of which more heat is lost from the skin surfaces than can be supplied by the rise in metabolic rate; the other, which is met shortly before death in severe intoxication and in the last hours of the paralytic stage, is the inability of the dog to maintain her metabolic rate at the ordinary level."

LYTTLE, New York.

COMPARISON OF A VIRUS OBTAINED BY KOBAYASHI FROM CASES OF EPIDEMIC ENCEPHALITIS WITH THE VIRUS OF RABIES. E. V. COWDRY, J. Exper. Med. **45**:799 (Sept.) 1927.

During an outbreak of a form of epidemic encephalitis in Japan, in 1924, Kobayashi isolated a "virus" from a single case. Cowdry, studying this virus at the Rockefeller Institute for Medical Research, concluded: "Since the symptoms produced in experimental animals by the encephalitic virus of Kobayashi and by the virus of rabies are similar, and are accompanied by lesions, which are indistinguishable, and since a cross immunity is demonstrable between the two viruses, the conclusion is advanced that the specimen of so-called encephalitis virus, isolated by Kobayashi, is in reality a specimen of rabic virus."

FREMONT-SMITH, Boston.

OBSERVATIONS WITH THE PRECIPITIN REACTION. LUDVIG HEKTOEN, J. Immunol. **14**:1, 1927.

The serum proteins, hemoglobin, and the specific precipitinogen in semen appear to be distinctive or specific for the species, and consequently presumably different chemically in each species, while the lens proteins, fibrinogen and thyroglobulin, appear to be more or less closely related in many distinct species. Of the latter group, the lens and casein have been the standard illustrations to which can now be added fibrinogen and thyroglobulin. That fibrinogen should disregard so completely the species limitations of the immune reactions of serum and its constituents as well as of hemoglobin is both surprising and interesting. In fibrinogen the blood reveals a much wider antigenic relationship than is ordinarily assumed to be the case. It is of special interest in connection with the fundamental problems of immune specificness and the production of antibodies that these nonspecies-specific antigens can evoke the formation of corresponding antibodies in the homologous species, at least under certain conditions. In view of this circumstance, may it not be well to subject to renewed experimental scrutiny the old assumption that species-specific antigens are incapable of provoking any antibody response in their own species?

AUTHOR'S SUMMARY.

EXPERIMENTAL IMMUNIZATION IN POLIOMYELITIS. W. LLOYD AYCOCK and J. R. KAGAN, J. Immunol. **14**:85, 1927.

It was found that attenuated virus was little less if not as pathogenic as active virus on subcutaneous injection in monkeys. The intracutaneous injection of active virus did not produce infection and appeared to protect monkeys from intracerebral injection of active virus to considerable degree. The blood

serum of monkeys injected by the intracutaneous method neutralizes poliomyelitic virus in vitro. The results support the hope that immunity to epidemic poliomyelitis may be produced without active symptoms.

ARCH. PATH.

THE DEGENERATION OF COMPLEMENT ON STORAGE AT SEVERAL TEMPERATURES. JOHN F. NORTON, BLAKE BARFIELD and I. S. FALK, J. Infect. Dis. **41**:39, 1927.

We have found no evidence of the regeneration of complement during storage over a period of forty-two days at temperatures of 37, 4 to —6, and —10 C. Complement stored at 37 C. deteriorated rapidly. The activity of both undiluted and diluted serum disappeared by the end of the third day of storage at this temperature. Both undiluted and diluted serums retained their original complementary activity for forty-eight hours at from 4 to 6 C. At the end of seven days the unit was found to be from two to three times as great as that of the fresh serum. Frozen serum kept at —10 C. retained its complementary power for two days, after which deterioration proceeded slowly. The undiluted serum could be used for about two weeks, the diluted serum for not more than from five to seven days.

Guinea-pig serum possessed the greatest complementary potency. Swine serum ranked second. Horse and rabbit serums could be used but were less satisfactory. Sheep serum failed to complement the hemolytic system studied, due to the absence of the complementary factor rather than to the presence of interfering substances.

The rate of deterioration of complementary activity could be expressed by use of a monomolecular equation, although it does not necessarily follow that the reaction is monomolecular.

The constants obtained with this equation were approximately the same for the four serums stored at any one temperature, showing that the rate of loss of complementary activity was nearly identical for guinea-pig, swine, horse and rabbit serums. This may be considered as evidence in favor of the view that the complementary factor is the same, regardless of its source.

THE POTENCY OF STORED PERTUSSIS VACCINES. LUCY MISHULOW, CAROLYN OLDENBUSCH and MARIE SCHOLL, J. Infect. Dis. **41**:169, 1927.

If the antigenic value of pertussis vaccines can be measured by their power to stimulate specific antibodies, and if the response in human beings is similar to that of rabbits to immunization with the vaccines, then properly stored vaccines remain antigenically potent for a considerable length of time, and the claim that pertussis vaccines lose about 75 per cent of their potency after storage for two or three months has not been substantiated. Eight different preparations, stored at from 8 to 10 C. and tested periodically, were highly potent at the end of four and one-half years.

AUTHORS' SUMMARY.

THE TITRATION OF SCARLET FEVER ANTITOXIN IN RABBITS. H. J. PARISH and C. C. OKELL, J. Path. & Bact. **30**:521, 1927.

By means of a test involving the protection of rabbits against the septicemia produced by the intravenous injection of the scarlet fever streptococcus, it has been found possible to distinguish efficient therapeutic serums from inefficient. The test has been used on a practical basis as a method of titrating antitoxin. Evidence is produced which strongly suggests that the antibody measured in the test is scarlet fever antitoxin and so far as it has been explored, the test has been found to run parallel with the usual methods of titrating antitoxin in human subjects.

AUTHOR'S SUMMARY.

BLOOD GROUPS IN CHILDREN, IN HEALTH AND DISEASE. A, PAGANI-CESA, Clinica Pediat. **9**:775 (Oct.) 1927.

The percentages of a class of Italian children divided into the various blood groups are as follows: group I, 46.1; group II, 32.3; group III, 11.5; group IV, 10. These percentages correspond to those obtained by other observers.

Various diseases do not alter the grouping, nor are members of the various groups susceptible to certain diseases.

ZAMBARANO, Northville, Mich.

INFLUENCE OF ULTRAVIOLET RAYS ON SERUM IRRADIATED IN VITRO: I. INORGANIC PHOSPHORUS AND $p_H$. G. FRONTALI, Riv. di. clin. pediat. **25**:661, 1927.

Blood from twelve persons (four rachitic children, two tuberculous children, one nephritic child, one undernourished child, one normal child and two normal adults) was analyzed for inorganic phosphorus before and after the blood had been irradiated in vitro; in six of the cases, the $p_H$ was determined before and after irradiation. In ten instances, the inorganic phosphate was increased (the average increase equaled 0.4 mg. per hundred cubic centimeters); in one (a normal child), it was unchanged, and in one (an undernourished infant), it was diminished. In each case, the $p_H$ rose (i. e., the blood became more alkaline).

HIGGINS, Boston.

THE FORMATION OF ANTIBODIES IN THE NURSLING AND THEIR THERAPEUTIC IMPORT. MARIO SCHTEINGART and PASCUAL R. CERVINI, Arch. Latino-Am. de Pediat. **21**:829, 1927.

Nursing infants of any age are capable of reacting to the parenteral introduction of vaccines by the production of agglutinins and specific antibodies. The debilitated infant does not differ from the normal in this respect. Every infant presents a threshold for the quantity of germs required for the production of positive reaction.

SCHLUTZ, Minneapolis.

THE PHYSICOCHEMICAL PROPERTIES OF BILIRUBIN IN BODY FLUIDS. E. FORRAI and R. SIVO, Biochem. Ztschr. **189**:162 (Oct.) 1927.

The authors wished to determine whether the difference between the direct and indirect van den Bergh reactions is explicable on the basis of a different physical state of the bilirubin in the serum. Many think that the direct reaction is due to a crystalloid form and the indirect to a colloidal state of the bilirubin. The authors used a Bechhold apparatus with a 4.5 per cent glacial acetic acid and collodium filter under an oxygen pressure of from 4 to 8 atmospheres. Ninety per cent of a prepared crystalloid solution of bilirubin passed through the filter. Normal serum, serum with increased bilirubin content and an indirect reaction, serum with a direct reaction and bile from the duodenum were tested. In none did the filtrate contain bilirubin. The bilirubin present in urine, however, passed through the filter. This indicated that fluids containing protein retain bilirubin even when the van den Bergh test is positive. Addition of a crystalloid solution of bilirubin to serum prevented the ultrafiltration of the bilirubin. The addition of gum arabic (a nonprotein colloid) also prevented the filtration of crystalloid bilirubin. The authors conclude that the bilirubin present in serums giving a direct van den Bergh reaction is bound by the colloids present.

THE EFFECT OF SPLEEN AND BONE MARROW EXTRACTS ON BLOOD REGENERATION AFTER PHLEBOTOMY. T. HONDA, Biochem. Ztschr. **190**:456 (Nov. 23) 1927.

Numerous investigators have concluded that the spleen has a restricting effect on the function of the bone marrow. After producing an anemia in rabbits by removing large quantities of blood, the author fed the animals 0.324 Gm. of dried bone marrow or dried spleen daily. The hemoglobin in the

control animals returned to normal in twelve days. The hemoglobin in animals fed bone marrow reached the normal level in nineteen days, and in those fed the dried spleen in twenty-six days.

BRAHDY, New York.

THE HISTOPATHOGENESIS OF PERTHES' DISEASE, ON THE BASIS OF AN EARLY CASE. KARL ROCKEMER, Frankfurt. Ztschr. f. path. **35**:1, 1927.

The histologic changes in an early case of Perthes' disease are described. There was an almost complete necrosis of the spongy bone of the head of the femur, although the articular cartilage was still supported by it. Furthermore, the spongy part of the head had been the site of a pathologic impression fracture. Evidence of repair could not be demonstrated in the early stage of the disease.

Rockemer points out that in this and a number of other cases of Perthes' disease the changes are in agreement with those seen in Köhler's disease of the head and of the second metatarsus. He attempts to decide the pathogenesis of Perthes' disease on the basis of the histologic picture, and thinks it probable that a disturbance of nutrition lies at the basis of the disease made possible by the special nutritive conditions for the head of the femur. The nutritive disturbance in most cases is brought about by trauma.

E. M. HALL.

PNEUMOCOCCUS PLANUS: ETIOLOGY OF INFLUENZAL DISEASES IN CHILDREN. A. ADAM, Jahrb. f. Kinderh. **112**:237 (May) 1926.

From his bacteriologic investigation of acute and chronic influenzal infections and their complications, Adam concluded that the pneumococcus is the most important agent of such infections in childhood. In blood-agar cultures of the nasal and pharyngeal secretion from 140 children of the first two years of life, there were 60 per cent pneumococci; of 84 children from 2 to 14 years, there were 51 per cent from 2 to 6 years and 33 per cent from 6 to 14 years, and only 2.5 per cent in 170 adults. The organism is pneumococcus group IV. It forms flat colonies, has marked fermenting power, and is designated by Adam as *Pneumococcus planus*. It seems to cause a typical children's disease, the etiology of which is not yet explained. Infants reacted to the specific vaccine much more weakly than did older persons. A sensitiveness to bacterial toxins does not seem to be dependent on infection or disposition.

PANCREATIC ATROPHY IN INFANCY AND IN CHILDHOOD. FRITZ GROSS, Jahrb. f. Kinderh. **112**:251 (May) 1926.

In twenty-seven necropsies of children between 6 days and 10 years of age, Gross examined the pancreas, in which he found deviation from the normal to be a frequent occurrence. Atrophy of the parenchyma is prominent among the severe injuries. Severe lesions of the islands were not encountered. In the lipoid examination four cases showed fine staining of the islands and separate tubules; one showed marked infiltration. In one case the pancreatic changes were probably responsible for the disease or cachexia of the child; in one, a teratoma caused pressure atrophy of the pancreas, but the remaining cases appeared as organic atrophies parallel with general wasting in the organism. The diseases especially disposing to this condition are those of the digestive tract and acute and chronic toxi-infectious processes.

PACE, Chicago.

FATS AND LIPOIDS IN CERTAIN ENDOCRINE ORGANS. H. P. GROSSMANN, Virchows Arch. f. path. Anat. **265**:137, 1927.

Grossmann examined for their content in fats and lipoids the parathyroids, the thyroid and the suprarenals in 100 necropsies of persons varying in age from 4 weeks to more than 70 years. He defines fat as stroma cells filled with

neutral fats, i. e., adipose tissue. Such tissue was present in all except six parathyroids, and in all but eleven thyroids. Adipose tissue cells occurred singly or in small groups in the cortex of 21 of 150 suprarenals. Intracellular lipoids, chiefly phosphatids and lecithin, were uniformly present within the parenchyma cells of the three organs examined. The presence of fat is believed to depend on the nutritional state of the individual organ. The lipoid content of the suprarenal cortex is held to have no relationship to that of any other organ, but to be dependent on the lipoid content of the blood which reaches the cortex. The lipoids of the cells of the parathyroids and the thyroid are the result of intracellular metabolism, and their amount is an expression of the metabolic activity of the cells of these organs.

O. T. SCHULTZ.

THE VALUE OF THE UROCHROMOGEN TEST IN CLINICAL MEDICINE. A SUMMARY OF ONE THOUSAND SEVEN HUNDRED AND SEVENTY-TWO TESTS. Y. Y. YING, National M. J. China **12**:453 (Dec.) 1926.

The urochromogen test, devised by Weisz, is known to be of value in the diagnosis and prognosis of tuberculosis and fevers of the enteric group. Ying made 1,772 separate tests which included 1,349 patients with different diseases (primary diagnosis) and 423 normal persons. He also studied the secondary diagnosis of each case carefully. The technic outlined by Webster was followed: "Place the suspected urine in a test tube and dilute with three times its volume of water. Mix well and add 3 drops of a 1:1,000 aqueous solution of potassium permanganate. Shake well. A yellow tint or an increase of the yellow tone of the urine indicates a positive reaction." The author found most of the positive reactions in typhoid, malaria and tuberculosis and he cites the percentages of these. He did not find that various drugs administered before the test was made influenced its specificity. The urochromogen test was uniformly negative in normal persons and has not been noted in diseases other than those already named.

THE INFLUENCE OF VARIOUS ALBUMINS ON THE PROPORTION OF SOME NITROGEN CONTAINING SUBSTANCES IN THE URINE OF THE INFANT. PE-SHEN CHANG, National M. J. China **12**:460 (Dec.) 1926.

This article is in German with a summary in English. The object of the investigation was to determine the correlation between the ingestion of different kinds of albumin and the proportion of urea, ammonia and uric acid in the urine. Three healthy infants, two on basic diets of butter-flour preparations (poor in casein) and the other on half milk porridge, had the following additions: (1) purée of liver; (2) casein and (3) vegetable albumin (from wheat embryo). The author gives his method of testing for total nitrogen, urea and uric acid. He found that infants excreted comparatively less urine than adults, but that they excreted high quantities of ammonia, uric acid and residual nitrogen. This observation is in accordance with former investigations on the subject. "If liver purée was given, the urea contents were higher (Ca 7 per cent more) than with the other two kinds of albumin. Just the reverse was the case with ammonia. Striking is the high excretion of urea. . . . The residual nitrogen is higher when casein or vegetable albumin are given than with liver albumin."

Charts showing the experiments are included.

LUCAS, San Francisco.

## Nutrition

STUDIES IN RICKETS. H. J. GERSTENBERGER, J. I. HARTMAN, JOHN D. NOURSE, NORMAN C. WETZEL and D. M. SMITH, Ann. Int. Med. **1**:305 (Nov.) 1927.

The basic cause of rickets is inadequate exposure to actinic rays. Cod liver oil cures rickets in a different manner than does the actinic ray. As proof of the first statement, the four following facts are offered:

1. Exposure to actinic rays cures rickets regardless of diet.

2. Undiluted cow's milk aids rather than prevents rickets.

3. Human milk does not protect against and will not cure rickets in human offspring even though the mother ingests potent cod liver oil.

4. Human milk from a mother adequately exposed to actinic rays cures active and nonhealing rickets in human infants.

Facts 3 and 4 are supported by experimental evidence in the article.

In proof of the second statement, the authors observe that clinical rickets occurs in that part of the osseous system which is growing most rapidly. In experiments with premature infants they found that when the gain in weight was slow, it took less cod liver oil per gram grain in weight per kilogram of body weight to cure rickets than when the gain in weight was rapid. A curve was constructed indicating the amount of cod liver oil per daily gram gain in weight per kilogram of body weight; this curve was used as a determinant in preventing rickets in seven premature and one full term infant for a period of from four to five months. The maximal daily average amount of cod liver oil was 0.71 cc., and the minimal amount was 0.1 cc. Only one infant developed evidence of mild rickets. The authors conclude, therefore, that rickets in rapidly growing premature infants can be prevented with cod liver oil, and that the amount of cod liver oil required is small and varies directly with the rate per gram gain in weight per kilogram of body weight.

GIBSON, Elkhart, Ind.

EXPERIMENTAL STUDIES CONCERNING THE RELATIONS BETWEEN THE SKIN AND VITAMINS. W. RICHTER, Arch. f. Dermat. u. Syph. **153**:697 (Oct. 23) 1927.

Six guinea-pigs were fed on a diet from which vitamin C was absent. All died with symptoms of scurvy in from ten to fifteen days. In six other guinea-pigs an area of about 2 cm. on the back was shaved. This was covered with a thick gauze dressing which was attached firmly to the surrounding skin except for a small window cut in its central portion. Into this window, the juice of spinach, extracted by pressure by a special method, was dropped four times a day, until the dressing was saturated. These animals received the same diet as the first six. In from ten to sixteen days, three died with manifestations of scurvy. The remaining three remained well over an observation period of three months. Another series of guinea-pigs were treated in the same manner, except that minerals (potassium, calcium, magnesium, sodium, combined with phosphorus, sulphur and hydrochloric acid, and traces of silicic acid) were added to the spinach juice. The mortality in this group was 30 per cent. In a further series of guinea-pigs, the treatment was the same except that the animals were fed on the vitamin-free diet for from two to six days before the spinach juice was applied to the skin. Regeneration set in within six days in 60 per cent. From the eighth day on, the mortality curve fell rapidly. The comparatively high mortality figures are explained by the complicating infections common in experimental animals. Richter draws the following conclusions: the skin is able to absorb vitamins. From the fact that the spinach juice expressed according to the method used by him contains only slight traces of chlorophyl and that its vitamin action was of so high a grade, it must be assumed that the vitamins are not bound to the chlorophyl alone. Since the addition of minerals to the spinach juice considerably improved the results, it appears that minerals have the property of activating vitamins. Regeneration from injury due to lack of vitamins is possible only up to a certain degree.

CARPENTER, Chicago.

THE EFFECT OF A MEAT DIET ON THE RESPIRATORY METABOLISM AFTER DIFFERENT TYPES OF DIET. T. HONDA, Biochem. Ztschr. **191**:34 (Dec. 8) 1927.

The specific dynamic action of meat was studied in rats which had been fed on various diets. After a diet of bread and milk, the specific dynamic

action of meat was observed. The average carbon dioxide elimination was increased 7.35 per cent and the oxygen intake, 3.58 per cent. This was further increased if cane sugar was added to the diet preceding the feeding of meat. Following a diet of bread, milk and sugar the feeding of meat resulted in an average rise in the carbon dioxide elimination of 13.74 per cent, in oxygen consumption, of 7.43 per cent and in calory production, of 7.85 per cent. A fat diet preceding the intake of meat practically eliminated the specific dynamic action. The author thinks that the liver plays an important rôle in the specific dynamic action of ingested meat. In one series the liver was infiltrated with fat, and in the other it contained an excess of glycogen.

BRAHDY, New York.

THE ABILITY OF THE INFANT'S PANCREAS TO DIGEST CELL NUCLEI. J. VON LUKACS, Monatschr. f. Kinderh. **37**:193, 1927.

Whether the infant's pancreas can digest cell nuclei has been a disputed problem. To settle this point, the author tried experiments both in vivo and in vitro. In the first set, he gave children scrapings of calf's liver contained in tiny sacks permeable to digestive juices, and later examined the stools. In the other set, he exposed small pieces of ground up liver to the action of pancreatic juice. The results of these experiments showed that during the first year of life, the infant's organism is unable to change the structure of animal cell nuclei. In the test tube extracts of adult pancreas are effective, while the infant's pancreas lacks the nucleus splitting property. It was found by chemical experiment, however, that purin bases can be extracted from cells without any change in nuclear morphology.

GERSTLEY, Chicago.

THE ANTIRACHITIC PRINCIPLE OF COD LIVER OIL. LEON A. TSCHERKES and I. I. LITWACK, Ztschr. f. Kinderh. **43**:403 (May) 1927.

The researches of Tscherkes and Litwack have established the fact that vitamin B is lacking in cod liver oil, and that if cod liver oil is an antirachitic agent it is not due to vitamin B, as von Reyher assumed, but to another substance which cannot be substituted by preparations containing only vitamin B.

PHYSIOLOGY OF DIGESTION IN INFANTS: I. THE DIGESTION OF FAT. ERNST FREUDENBERG, Ztschr. f. Kinderh. **43**:437 (May) 1927.

At the end of the third or fourth hour of digestion, the gastric juice of infants contains a kinase called the lipo-kinase of the stomach which activates the lipase of human milk. Lipo-kinase is thermolabile, soluble in alcohol, is not dialyzable and is adsorbed from cellulose at neutrality but not from glass-wool. It has no relation to rennet and is lacking in the saliva. In pathologic cases, especially in recent dyspeptic conditions, there may be a diminution or absolute lack of lipo-kinase.

The lipase of human milk is also activated by biliary acids and can be reactivated in the intestine by bile. It tolerates a temperature of 37 degrees at $p_H$ 5 without injury. As a ferment, it is much more active than gastric lipase, for it splits, activated by lipo-kinase, from 30 to 40 per cent of the milk fat in two hours. Fortified by duodenal juices, it splits the fat of human milk almost quantitatively in two hours. Splitting of the fat of human milk in the stomach does not take place at $p_H$ 5, but splitting of the lower fatty acid of cows' milk does take place. At $p_H$ 6, bile increases the turn-over of the lipase of human milk markedly. The quality of protecting the higher fatty acids belongs to the protein bodies of whey, at an acid reaction before precipitation, and this protection is increased by bile.

By the use of 2 per cent watery solution of hematoxylin a differentiating reaction is given between human and cows' milk and between raw and cooked cows' milk, which is dependent on the content of casein.

EMERSON, Boston.

STUDY OF THE CONNECTION BETWEEN CHLORINE CONTENT OF THE BLOOD AND BLOOD PLASMA AND GASTRIC SECRETION. F. ROHRBÖCK and J. ISTVÁUCSICS, Orvosi hetil. **44**:1260, 1927.

Chlorine determinations on the blood do not seem to show any parallelism with the gastric secretion in older children. This is in contrast to conditions in infants and adults. The explanation may be that at this age extreme pathologic conditions are seldom found. The most reliable method of examination at this age seems to be the simple test breakfast.

ZENTAY, St. Louis.

## Prenatal Conditions

CONGENITAL HYPERTROPHY, INVOLVING THE RIGHT SIDE OF A FEMALE AGED SIX. E. G. WAKEFIELD, Ann. Int. Med. **1**:292 (Nov.) 1927.

Wakefield reports the case of a girl, aged 6 years, with congenital hemihypertrophy involving the right half of the body. In his discussion, he states that congenital hypertrophy is more frequent than the literature indicates. Twice as many cases have been reported in the last thirty years as during the sixty years preceding, due to more frequent recognition rather than increased incidence.

GIBSON, Elkhart, Ind.

ABNORMALLY LARGE FORAMINA PARIETALIA. DAVID M. GREIG, Edinburgh M. J. **34**:629 (Nov.) 1927.

Greig refers to rare instances of unusually large parietal foramina and gives numerous illustrations. Normal foramina are found in a large percentage of skulls. They give exit to branches of the middle meningeal and occipital arteries and veins. Foramina may be present on both sides or absent on one or both sides. It is the large size of certain foramina which makes them abnormal. Greig believes that the abnormality is a dysplasia dependent on incomplete local and general development of the cranial bones, congenital in origin. The condition is seldom recognized in life, though it was found in one child at operation and was not due to craniotabes.

ON THE PERSISTENCE OF COMPLETE WOLFFIAN DUCTS IN FEMALES. DAVID M. GREIG and GERTRUDE HERZFELD, Edinburgh M. J. **34**:701 (Dec.) 1927.

Herzfeld operated in two rare cases, the patients being girls, aged 3 and 5 years, respectively, who since infancy had had a vulvar discharge of sterile pus from a small sinus opening on the hymen at its superior attachment to the vagina, or on the floor of the urethral meatus. A bismuth compound had been injected into the opening, and a tortuous, tubelike shadow was revealed passing up over the brim of the pelvis. The tumors were removed successfully, and the children were cured.

The authors discuss the anatomy and development of the wolffian ducts and their persistence. These structures must be differentiated clinically from so-called duplication of ureters, but they are entirely different because they do not have any connection with the kidney, ureter or bladder. In the male, the müllerian duct may be persistent and mistaken for an accessory ureter.

The subject is discussed at length and the literature completely cited.

NEFF, Kansas City.

CONGENITAL DEFECTS OF THE MUSCLES OF THE CHEST. ERNEST MELZNER, Arch. f. klin. Chir. **144**:131, 1927.

The author reports two cases of congenital absence of the right pectoralis major and minor muscles. In one patient, a boy, aged 9 years, webbed fingers of the right hand involving the second and third and the fourth and fifth, were associated with the condition. On the affected side, the shoulder as well

as the nipple was elevated and the intercostal spaces easily visible. Often a mild torticollis is present, the head being directed toward the normal side. The clavicle and scapula usually are smaller than on the normal side.

The other patient was a boy, aged 19 years, who fell and hurt his shoulder. Three days later two doctors were called, who diagnosed the condition as broken ribs on account of the depression, not recognizing the defect in the muscles. When the author saw this patient, he at once recognized the defect and called the attention of the patient to it, as he had never noticed it previously.

The theories for this condition present two possible causes. One is possible pressure against that part of the embryo by an arm, the head, or even a uterine fibroid, and these must have occurred during the first one or two months of intra-uterine life. The other cause may be failure either of growth or of primary germ layer, or even absence of nerves to that part.

CONGENITAL DEFECTS OF THE SPINE. GUSTAV DREHMANN, Beitr. z. klin. Chir. **139**:191, 1927.

The author calls attention to the infrequency of congenital defects of the spine before the advent of the roentgen ray. He reports a case of congenital deformity of the spine in a boy, aged 15 years, who had a numerical reduction of the vertebrae with lack of differentiation into the individual vertebrae and with synostosis of the vertebrae and of many of the ribs.

In a second case, a child, aged 2 years, with a marked kyphoscoliosis of the lumbar spine, had only a rudimentary first lumbar vertebra. In a third case, a boy, aged 1 year, with clubfoot and some paresis of the muscles, had a marked defect of the sacrum, only three lumbar vertebrae, and a rudimentary fourth lumbar, vertebrae lying in the place of the sacrum.

OBERHELMAN, Chicago.

CASE OF ANONYCHIA CONGENITA WITH LOSS OF PHALANGES. A. DOMBROWSKI, München. med. Wchnschr. **73**:2168 (Dec. 17) 1926.

The author reports a case of anonychia congenita in a patient, aged 34 years. The nails on the toes were absent. A peculiar feature was that on the second, third and fourth toes of the left foot and on the second and third toes of the right foot the middle and end phalanges were absent. A hereditary influence could not be established.

AMBERG, Rochester, Minn.

## Diseases of the New-Born

MEDIASTINAL EMPHYSEMA IN THE NEW-BORN. K. KAISER and G. SCHRADER, Monatschr. f. Geburtsh. u. Gynäk. **77**:205 (Oct.) 1927.

The details of the labor are given. The head was poorly engaged when the membranes ruptured. Twice during labor the fetal heart rate dropped suddenly, and material the color of meconium was expelled. A cyanotic infant, weighing 2,500 Gm. and 49 cm. long was delivered spontaneously and died in twelve hours. Artificial respiration was not given. Autopsy revealed air in the anterior and posterior mediastinum, pericardial sac and left auricle and ventricle, a few air bubbles in the coronary arteries, and several small cerebral hemorrhages. The authors feel that the rupture of the membranes resulted in a sudden change in pressure which caused the cerebral hemorrhages, one of which injured the respiratory center. This resulted in the two intra-uterine attacks of asphyxia, indicated by the sudden drop in fetal heart rate and the passage of meconium. The forced breathing of the asphyxiated new-born infant ruptured the lung tissue. The authors emphasize that cerebral hemorrhages are not always due to instrumentation, and that rupture of the lung tissue is not necessarily due to vigorous methods of artificial respiration.

BRAHDY, New York.

TWO UNUSUAL SKIN DISEASES IN THE NEW-BORN. EMIL FLUSSER, Monatschr. f. Kinderh. **37**:205, 1927.

Flusser describes an infant with congenital syphilis in whom the entire skin showed a complete infiltration which he diagnosed as diffuse syphilitic infiltration of the skin. The child lived six days.

Another infant with aphthous stomatitis showed an excoriated area running from the bottom of the chin over the skin to the upper thorax. This was covered with a green-gray membrane. Cultures for diphtheria were negative. The author believes this to be an aphthous dermatitis.

In discussing aphthous stomatitis, he emphasizes that it is a specific infectious disease. It must be differentiated from aphthae due to other causes, as for instance, a chronic aphthous infection which may be due to syphilis or to Vincent's angina or to disturbed digestion. He reports two interesting cases, in one of which aphthae were associated with meningitis, and in another with an attack of paratyphoid.

GERSTLEY, Chicago.

RELATIONS BETWEEN ICTERUS NEONATORUM AND WATER ECONOMY. ELLY SANDER, Ztschr. f. Kinderh. **43**:434 (May) 1927.

In different forms of icterus in adults, Pollitzer and Stolz found disturbance of the water economy, which apparently could be traced to hyperirritability of the blockading devices of the liver already demonstrated by Pick. They showed that the disturbance is functional because it could be controlled by pharmacologic means. The effect of this active throttling in the liver is, on the one hand, a retardation of diuresis and, on the other, an increase of residual water in the organism which can be demonstrated by the loss of weight that is obtainable by the use of merbaphen in twenty-four hours.

In 1,500 new-born infants, Sander found that the ones with much icterus, on the average, showed the lowest and the nonicteric ones the greatest weight at birth. The physiologic loss of weight is absolute and greatest in the severely icteric infants. This higher water loss of the organism is not conditioned in part by a lesser intake of fluid; on the contrary, it stands much more in relation to a greater intake of fluid. The icterus of the new-born infant prolongs the physiologic loss of weight and slows its recovery. Icterus neonatorum influences the water economy of the new-born organism in the sense of an increased water output.

EMERSON, Boston.

## Acute Infections

SURGICAL SCARLET FEVER. T. BANFORD JONES and LAWRENCE D. KOHN, Arch. Surg. **16**:139, 1928.

A child, aged 15 years, had been exposed to scarlet fever in his own household and, shortly after quarantine had been lifted, developed cellulitis of the right leg following an abrasion of the right great toe.

Eighteen hours after admission to the hospital, a sharply positive Dick test was obtained, and a positive blanching test with scarlet fever antitoxin was obtained at the edge of the lesion on the right shin.

The authors describe the isolation of *Streptococcus scarlatinae* from the initial lesion on the toe, and consider the case one of scarlet fever in which the causative organism invaded the toe and produced its toxin there and in the leg instead of the usual pharyngeal location.

GEORGE H. JACKSON.

NATURE OF THE TOXIC MOIETY OF STREPTOCOCCUS SCARLATINAE. CHARLES W. DUVAL and R. J. HIBBARD, J. Exper. Med. **46**:379, 1927.

Further evidence is presented in support of the view that the poisonous substance of the scarlatinal streptococcus is derived from the bacterial cell through dissolution of its plasm.

AUTHORS' SUMMARY.

REMEDIES RECENTLY INTRODUCED IN THE THERAPY OF AMOEBIASIS. OTTO WILLNER, Medicine **6**:341 (Sept.) 1927.

The prevalence of amebiasis is stressed, and attention is drawn particularly to the difficulty of diagnosis by clinical and laboratory means. The clinical manifestations vary from frank, acute, violent symptoms to a chronic stage in which the only tangible evidence of disease is that a patient does not "feel well." There is lack of standardization of methods of investigation, so that statistics are not absolutely reliable. Emetine still remains the most effective therapeutic agent in the acute cases, but the author thinks that the recent tendency to increase the dosage of emetine not only is not effective but is fraught with considerable danger.

Yatren is the subject of special consideration in this study. Yatren is a German proprietary substance. The chemical composition is stated to be:

| | | |
|---|---|---|
| Iodine ..... | 5 parts | Containing 28 per cent of iodine |
| Oxychinoline ..... | 8 parts | |
| Sodium sulphate... | 7 parts | |

Sodium bicarbonate, 20 per cent, is added to increase its solubility. The bactericidal potency of this drug on many organisms and in various dilutions in vitro is given. Administration of this drug is as follows: When it is used as an enema, spectacular benefit has resulted in severe chronic forms of the disease which had resisted other kinds of treatment. The solution recommended is from 2.5 to 1 per cent. Stronger solutions are apt to produce marked colonic irritation.

By mouth yatren has usually been given in wafers or gelatin capsules of 0.5 Gm., from four to six times a day, or in four pills of 0.25 Gm. each, three times a day. Its administration by mouth is usually followed by diarrhea, which subsides from twelve to twenty-four hours after the drug is discontinued. Alternate treatment per os and per rectum has been found practicable. Treatment must be modified with the tolerance of the patient. In the Peking Union Medical College, Peking, China, the following routine is employed: on the first, third, fifth and seventh days 0.5 Gm. of yatren is given by mouth six times a day. On the second, fourth, sixth, tenth, fourteenth and twenty-first days, an enema containing 3 Gm. of yatren in 200 cc. of distilled water is given, after a cleansing enema which is retained for several hours. This is called one course of yatren. The course is repeated if indication arises. An increase of diarrhea or colic is an indication to interrupt the treatment for a day and to try smaller doses if the symptoms recur. Some patients may be treated as ambulatory cases, but the best results are found after hospitalization for seven days. Special diet may or may not be prescribed.

For children, solutions of yatren have been used in doses roughly proportioned to their body weight. It is said to be well tolerated and effective. The results are said to be uniformly good and sometimes spectacular in chronic cases. It is difficult and often impossible to state when a cure is effected. The combination of treatment with yatren and emetine sometimes accompanied by conservative surgical treatment has been effective in a few cases of abscess of the liver.

"It is not surprising that occasional cases of sprue have been benefited by yatren, for amebiasis is not rarely disguised under this clinical picture."

Many cases have been reported in which treatment with yatren was entirely unsatisfactory, and opinions differ widely as to its effectiveness. The arsenical preparations are discussed, and reference given to work with them which has been reported as satisfactory. "Yatren provides us with the therapeutic test involving no risk."

ROYSTER, University, Va.

AVOIDING SHOCKLIKE EFFECTS IN TREATMENT OF SCARLET FEVER WITH CONVALESCENT SERUM. P. BODE, Jahrb. f. Kinderh. **114**:31 (Sept.) 1926.

Shock observed with intravenous administration of scarlet fever convalescent serum usually occurred from about twenty to sixty minutes after injection and

manifested itself in the less severe forms by a chill, rise in temperature, cyanosis and slight symptoms of collapse, and in the most severe forms by chill, cyanosis, high temperature, circulatory weakness and loss of consciousness, which symptoms led to death. The cause of this acute intoxication is not clear, but in some cases there seemed to be a primary incompatibility between the donor's serum and the organism of the recipient, a manifestation parallel to serum disease. In another portion of the cases, there seemed to be an anaphylatoxin action. In these, the serum of previously treated donors was used. In order to avoid shock phenomena, it is recommended that previously treated persons should not be used as serum donors and that intravenous use of scarlet fever convalescent serum be renounced and that intramuscular application be adopted. The use of large doses (from 40 to 100 cc.) of the scarlet fever convalescent serum is absolutely necessary.

WHOOPING COUGH ECLAMPSIA. ERICH HÄSSLER, Jahrb. f. Kinderh. **114**:376 (Dec.) 1926.

Seven cases are presented by Hässler. In an infant, aged 8 months, a latent spasmophilia became manifest during an attack of whooping cough and led to general convulsions. In a child, aged 5 years, antispasmophile treatment brought improvement immediately and finally cure. Lumbar puncture obtained improvement in the third case. In the sixth case, there was improvement without any special antispasmophile treatment. The vomiting was always most difficult to combat. In the last months, the author has used strontium in the form of a strontium chloride urea preparation. The children receive two or three or more intramuscular injections separated by intervals of a few days. Forty children, among them thirty-four infants, were treated. One infant, long underfed, died from exhaustion, and nine contracted pneumonia and died. Treatment had a favorable effect in twenty-six of the thirty remaining cases.

PACE, Chicago.

HISTOLOGY OF DEAFMUTISM AFTER SCARLET FEVER MENINGITIS. GERALD RUEMBLI, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:195 (Aug. 27) 1927.

The author describes the histologic changes in two cases of deafmutism which occurred after scarlet fever. The middle ear was normal in both instances. The two patients showed the symptoms of deafmutism caused by meningitis. Therefore, the conclusion was permissible that scarlet fever meningitis caused the deafmutism at a time when a malignant epidemic of scarlet fever occurred. The osseous obliteration of the labyrinth corresponded with the pictures observed in deafmutism after cerebrospinal meningitis, meningitis after measles, meningotyphoid fever, etc. The first patient, a woman, aged 71, had been admitted to the clinic, Oct. 6, 1920, suffering from a diffuse bronchitis, and died on October 17. At the age of 3 years, the patient had had meningitis after scarlet fever and had lost her hearing entirely. The second patient, a man, aged 58 was admitted to the surgical clinic on May 8, 1920, on account of tuberculosis of the ileocecum. He died, October 15, with the symptoms of thrombosis of the vena cava which was verified by necropsy. At the age of 9, the patient had become totally deaf following scarlet fever.

AMBERG, Detroit.

RESEARCHES ON SCARLET FEVER. E. GORTER, M. DE KORTE and J. MUNK, Acta Paediat. **6**:383, 1927.

The authors review the recent advances in the immunology of scarlet fever and give their own observations in this field. The Dick test, the Schultz-Charlton extinction phenomenon, the effect of antiscarlatinal serum and the prevention of the disease by immunization are discussed at some length.

*The Dick Test.*—With regard to the Dick test, the authors conclude that it is not an absolute criterion of immunity. A negative Dick test means immunity in nearly every instance, but there are a few exceptions to this. Thus the authors have noted that measles may temporarily abolish the Dick reaction. That the patient is not immune at this time is shown by the fact that his serum is incapable of neutralizing *Streptococcus scarlatinae* toxin. Conversely, a positive Dick test does not necessarily indicate susceptibility to scarlet fever: a considerable proportion of persons giving positive reactions to the Dick test will not contract the disease, probably because their immunity resides in tissues other than the skin. On the whole the Dick test is, however, the most useful indication of susceptibility which exists at present. It is probably more accurate than the toxin-neutralizing power of the serum. An attack of scarlet fever renders the Dick test negative in 93 per cent of the cases.

*The Blanching Test.*—In agreement with most observers, the authors find that this is a highly specific reaction for scarlet fever rashes, given only by the serums of persons giving a negative reaction to the Dick test. Some serums are more effective than others, probably because of differences in their antitoxin content.

*Antiscarlatinal Serum.*—Horse serum was used prepared according to the Dick method. No effect was noted when the serum was given after the fifth day. When it was given early, some benefit was noted, but this appeared to be offset by the severe serum sickness which occurred with great regularity. The authors have therefore abandoned the use of serum.

*Preventive Inoculations.*—By suitable injections of toxin the large majority of persons giving positive reactions to the Dick test can be rendered negative to the test. Reactions with symptoms of scarlet fever are often obtained, such as vomiting, fever, skin rash, sore throat and even the strawberry tongue.

THE DICK REACTION IN CHILDHOOD. NILS MALMBERG and G. JACOBSON, Acta Paediat. **6**:442, 1927.

From a study of more than 1,200 children, the authors conclude that:

1. The percentage of children giving positive reaction to the Dick test varies considerably with age. During the first few months of life, only from 15 to 20 per cent react positively. Between the ages of 2 and 5 years, a much larger percentage is found to give positive reactions: from 65 to 80 per cent. After this there is a gradual decline, and at the age of puberty only 25 or 30 per cent are found to react positively.

2. Following scarlatina, 89 per cent of the children gave negative reactions to the Dick test.

3. Pseudoreactions are more common in children hypersensitive to tuberculin.

4. Spontaneous alterations in the Dick reaction of patients were occasionally encountered. Rarely did a positive Dick reaction become negative but, conversely, a negative reaction found positive on retesting was not so rare. On this account, the authors believe that persons giving negative Dick reactions should be retested at least once.

EXPERIENCES WITH ANTITOXIC SCARLET FEVER SERUM. I. JUNDELL and N. LÖNBERG, Acta Paediat. **6**:463, 1927.

The serum was prepared by a method somewhat similar to that used by the Dicks. It was prepared from a single strain of streptococci isolated from a patient with scarlet fever. In a series of seventeen cases, this serum seemed of definite benefit on the disease itself, but its effect on complications was open to question.

The authors are now employing a polyvalent serum.

HOLT, Baltimore.

ENCEPHALITIS AFTER VACCINATION. D. WIERSMA, Acta Psychiat. et Neurol. **2**:167, 1927.

The author, in reporting eleven cases of encephalitis after vaccination, calls attention to the fact that sixty or eighty cases have occurred. There was considerable difference in opinion, however, among the investigators who described these cases as to the nature of the disease. Some believed it to be ordinary epidemic encephalitis; others reported varying symptomatology. The mortality was around 70 per cent.

The cases described by Wiersma developed from nine to nineteen days after vaccination. The first symptoms were weariness and drowsiness, but soon high fever, convulsions, paralyses and Kernig's sign occurred. Trismus was noted in some cases; the Babinski sign was almost always present, and ocular paralyses were not observed. The patients lay in coma rather than in sleep. Sequelae were not observed in the patients that recovered.

There is also some disagreement as to the anatomic aspect, since Lucksch described conditions typical of epidemic encephalitis, whereas Bastiaanse noted characteristic differences. "The alterations in encephalitis after vaccination are localized mainly to the white substance," and the basal ganglia and locus niger are spared. The pathogenesis of the disease is not clear. It was thought by some that the vaccine virus was contaminated; by others, that the patients harbored the virus of encephalitis, which became active following the lowered immunity resulting from the induced infection. It may be that some new virus activated by the vaccine virus is yet to be discovered.

FREEMAN, Washington, D. C.

## Chronic Infections

LATE CONGENITAL SYPHILITIC GUMMATOUS RENAL APLASIA. L. CHATELLIER and C. LAURENTIER, Ann. de dermat. et syph. **8**:608 (Nov.) 1927.

The condition resulted fatally in a girl, aged 18. The observations at necropsy are given, and the case is completely discussed, with stress on the similarity between congenital and acquired syphilis.

ANTIGENIC SUBSTANCES AND THE AUTOLACRIMO REACTION IN TUBERCULOSIS. G. DE SIMONE, Clin. Pediat. **9**:531 (July) 1927.

In a series of studies the author found that in tuberculous persons antigenic substances may be found in the lacrimal secretion as well as in the urine. These substances are not present in the lacrimal fluid of normal persons nor in tuberculous subjects whose lesions are inactive. These substances are quantitatively proportionate to the activity of the tuberculous lesions.

The presence of these antigenic bodies was demonstrated by intradermal tests made with the lacrimal fluid obtained from tuberculous patients and injected into allergic persons or into the patient himself.

The tests were carried out in thirty-four patients, and the Wildbolz auto-urine test was made simultaneously for comparison. The results of the two tests coincided in the majority of cases.

Although the technic of the autolacrimo reaction is much easier than the Wildbolz test, there is more room for doubt in its interpretation.

The author concludes that the auto-urine test is much more reliable for practical work.

ZAMBARANO, Northville, Mich.

THE VALUE OF THE SEDIMENTATION TEST IN SURGICAL TUBERCULOSIS IN CHILDREN. FRANZ BIER, Arch. f. klin. Chir. **144**:432, 1927.

The author applied the sedimentation test in 202 children with surgical tuberculosis, using the method of Westergren of just one hour as a period of sedimentation and on a basis of a millimeter measure. He attempted by this

method to determine the degree of tuberculous involvement as well as the location. In his series of 202 children, 21 had a normal or practically normal reaction, the reading being between 1 and 4 mm. In a second group of 59, the reading was from 8 to 15 mm. and in a third group of 82, from 15 to 40 mm. In a fourth group of 36, the reading was between 40 and 100 and in 4, over 100. The majority of children with tuberculosis of the hip joint had a high reading, as did those with renal tuberculosis. The type and degree of surgical tuberculosis is not always indicated, as one patient whose surgical lesion had been healed for six months had renal tuberculosis and a reading of 125 mm. Patients with the severest surgical tuberculosis are not always those that give the highest reading. However, in conditions in which the readings are consistently high, one must consider renal tuberculosis, whether the lesion is small or large. Likewise, any intercurrent infection will raise the reading. To be of value, this test must be done carefully, and the author gives it significance only from the standpoint of prognosis, for in all the patients with healed tuberculosis the reading returned to normal.

OBERHELMAN, Chicago.

TREATMENT OF TUBERCULOSIS IN CHILDREN. A. CZERNY, Jahrb. f. Kinderh. **114**:277 (Nov.) 1926.

Czerny has used protein therapy in tuberculous children for eight years and is convinced that, apart from its limiting the extension, it favorably influences the course of the disease. He first chose horse serum, later donkey serum and recently has been using ox serum. The horse serum was blank, but the other two serums were from treated animals. Frequently after subcutaneous injection of the serum from animals that had been injected with tubercle bacilli, severe skin reactions appeared in the children and necessitated the use of small doses (from 0.5 to 1.0 cc.) and an interval between them longer than twenty-four hours. As satisfactory as protein therapy has been, it does not cure every form of tuberculosis, nor does it cure quickly enough to eliminate the possibility of spontaneous cure.

EXPERIENCES WITH THE MEINICKE MICROREACTION. EDMUND KOVÁCS, Jahrb. f. Kinderh. **114**:308 (Nov.) 1926.

Dohnal recently described a microreaction for the diagnosis of syphilis which is based on the reaction of Meinicke. Only one drop of blood is necessary. This is placed in a capillary tube and allowed to coagulate. After centrifugalization, the serum is put in a leukocyte counting pipet with Meinicke's organ extract in a dilution of 1:11. After standing for one hour, the preparation is examined with the dark field. Dohnal and Meinicke consider this reaction more valuable than the Wassermann reaction. Kovács compared the results of the Meinicke microreaction and the Wassermann reaction in serums from 520 patients. The Meinicke microreaction agreed with the Wassermann reaction in 88.7 per cent and differed in 11.3 per cent of the cases. In the differing serums, the Meinicke microreaction was positive and the Wassermann reaction negative in 48 cases. Most of the instances of deviation were in patients with latent syphilis and in those being treated for syphilis. The author considers this reaction as a useful serologic method. In certain cases, it may supplant the Wassermann reaction.

PACE, Chicago.

CONGENITAL SYPHILIS OF THE LIVER WITH FEVER IN A FOURTEEN YEAR OLD BOY. W. MIKULOWSKI, Monatschr. f. Kinderh. **37**:218, 1927.

Mikulowski reports a chronic febrile disease associated with tumor of the liver in a boy who was being treated for tuberculosis. The condition was diagnosed as hepatitis leutica; antisyphilitic treatment was instituted, and a cure was effected.

GERSTLEY, Chicago.

LIPOID SOLUBLE BISMUTH IN TREATMENT OF CONGENITAL SYPHILIS. E. TÜDÖS, Wien. klin. Wchnschr. **40**:1510 (Dec. 1) 1927.

Tüdös treated congenital syphilis with a lecithin containing iodine-quinine-bismuth compound. It is claimed for this preparation that it is soluble in the body lipoids, slowly absorbable and nontoxic. Its effective dosage is said to be from one fifth to one sixth of that of other bismuth preparations.

Twenty-eight children, the majority of which were infants, comprised the series. The iodine-quinine-bismuth compound was used alone in twelve of these. Sixteen children were treated by a combined method in which, at three to four day intervals, two doses of the compound (from 0.2 to 0.3 cc. intramuscularly) were alternated with one injection of neoarsphenamine (from 1.5 to 2.5 cc. per kilogram of body weight) for a period of three weeks. All of the patients gained weight, and their syphilitic symptoms were promptly and markedly improved. Toxic effects due to bismuth were not observed. The effect on the Wassermann reaction was not uniform. Several positive reactions were slightly influenced or were not influenced at all; others became weakly positive, or negative, following one or two courses of treatment. Later, some of these returned to positive. The author concluded that a more rapid alleviation of symptoms was to be observed as the result of the iodine-quinine-bismuth compound-neoarsphenamine method than by bismuth used alone.

VACCINATION OF THE NEW-BORN AGAINST TUBERCULOSIS. A. CALMETTE, Wien. klin. Wchnschr. **41**:14 (Jan. 5) 1928.

This article is an answer to Nobel's recent charge that he has found Calmette's modified bovine tubercle bacillus (B C G) sufficiently virulent to kill inoculated guinea-pigs. For this reason, Nobel has advised against the general use of B C G for immunizing infants against tuberculous infection. Calmette repeats his previous assertion that B C G causes, at most, local abscesses or lesions in the lymph glands which subsequently heal spontaneously. He is of the opinion that there was an error in Nobel's experiments on the effect of B C G in guinea-pigs.

The author presents some new statistics on the results obtained with his vaccine. Of 1,891 vaccinated children who remained in contact with tuberculous relatives from one to three and one-half years, only five (0.2 per cent) died of tuberculosis. The mortality from tuberculosis in a group of 3,808 children under observation for less than one year after vaccination was 0.9 per cent. The general mortality in this group was 4.2 per cent as opposed to the general mortality in France of 8.5 per cent in the first year of life.

Calmette is opposed to subcutaneous injection of minimal doses of B C G in persons who give negative reactions to tuberculin, because he believes that the tuberculin rection denotes the presence of the disease only in those cases in which the lymph glands are already involved.

OLMSTED, Seattle.

DIFFICULTIES IN THE PRACTICAL EVALUATION OF THE TUBERCULIN REACTION. J. DUKEN, Ztschr. f. Kinderh. **43**:331 (May) 1927.

Positive tuberculin reactions in children who afterward have not shown, by the most painstaking examination at autopsy, signs of tuberculosis, are causing doubts as to the value of this reaction. It is Duken's opinion that the intracutaneous test is too sensitive and needs further research to make its interpretation perfect. It seems to him that in an infection with tuberculosis, when a positive Moro test follows a positive intracutaneous test, the test is more reliable and is to be given, then, a special significance.

PAPULOANNULAR TUBERCULID. J. DUKEN, Ztschr. f. Kinderh. **43**:335 (May) 1927.

Duken describes an infant, aged 6 months, who was infected with tuberculosis which was advanced enough to cause a cavity the size of a walnut in the lung. Nevertheless, at the age of 2½ years, the infant was in good general

condition, without demonstrable signs of the cavity. At the height of the disease, when the child was 11 months old, some peculiar spots appeared on the skin, without pain or itching. They occurred singly and in groups, were papulo-annular in character and were from 5 to 8 mm. wide. They were circular, with a slightly raised edge made up of small, hard nodules lying close together, and with a depressed center which was paler than the border. The general color was pale red with a slightly bluish shade. The exanthem gradually faded, became golden, then brownish, and completely disappeared in four weeks. Biopsy showed a nonspecific cell infiltration and no acid-fast bacilli. It seemed probable, however, that the condition of the skin was caused by tuberculosis. A second case was a perfect reproduction of the first, the skin lesions appearing on the site of the Moro reaction. An eruption like this has not been described in the literature and is a new form of manifestation of tuberculosis in the skin, to which the author gives the name of papulo-annular tuberculid.

THE INFLUENCE OF WHOOPING COUGH ON IMMUNITY TO TUBERCULOSIS. FRITZ MOSES, Ztschr. f. Kinderh. **43**:398 (May) 1927.

Whooping cough did not have a direct influence on the severe tuberculosis of the bones and joints observed in the institution supervised by Moses. It must be said that the children there were under unusually favorable surroundings and had been receiving fresh air and heliotherapy for months before infection with pertussis. Nevertheless, the author's results seem to show that in pertussis there is no diminution of immunity to tuberculosis. It should not be denied that whooping cough often influences pulmonary tuberculosis in infants and children unfavorably, but it is not true that immunologic processes play a real part therein.

PERIOMPHALITIS AND PERFORATION THROUGH THE UMBILICUS IN TUBERCULOUS PERITONITIS. WILFRIED COHN, Ztschr. f. Kinderh. **43**:411 (May) 1927.

Periomphalitis in tuberculous peritonitis is the expression of inflammatory and adhesive processes in the parietal peritoneum in the immediate neighborhood of the umbilicus. It is caused by (1) the sowing of tubercles on the parietal peritoneum in processes principally exudative; (2) fibrous adhesions between the omentum, intestine or mesentery on the one hand and the umbilical region on the other; (3) a superficial growing together of convolutions of intestine, omentum and lymph nodes with the parietal peritoneum near the navel, which, by direct adhesions of these organs, may cause a perforation of caseous lymph nodes or of a coil of intestine through the navel. Further, by the breaking down of a caseous tubercle or lymph node in an encapsulated peritoneal cavity, formation of a retro-umbilical abscess may occur and a perforation of this cavity into the intestine or a perforation of an ulcerating coil of intestine into the encapsulated retro-umbilical cavity may take place. Finally, a rupture of this type of cavity, which is filled with caseous and fecal contents, may occur through the navel externally. Simultaneous processes such as these may occur in histologically similar places in the abdominal wall; for example, in the scars of laparotomies. The prognosis of periomphalitis depends on the processes excited in the peritoneum. In the periumbilical sowing of tubercles over the parietal peritoneum, in adhesive peritonitis not leading to the formation of cavities (the more frequent cases), recession may occur, even if a secreting fistula or an intestinal fistula has appeared. However, if a larger cavity, which is in constant communication with the intestine and contains pus and blood exists, whether it has or has not been perforated externally, the retrogression of the infiltration is not to be expected. For the prognosis of the individual case, the course of the umbilical inflammation is without significance. This depends much more on the peritonitis as a whole. The periumbilical changes and the umbilical inflammation that is also present may heal, while at the same time or later other regions may be attacked. It is the condition of these which governs the outcome of the disease.

CONGENITAL SYPHILITIC CHANGES IN THE MILK TEETH. HANS WIMBERGER, Ztschr. f. Kinderh. **43**:429 (May) 1927.

In the milk teeth of congenitally syphilitic infants destruction limited to the upper incisors is often found, the other teeth remaining intact, a condition which can be traced to changes in the gum in the intermaxillary region just as with hutchinsonian teeth in the second dentition. The total destruction of the central superior incisors with severe circular caries of the external ones and an isolated complete destruction of the four upper incisors are characteristic examples in Wimberger's series of cases. It appears, then, that the generally accepted view that congenital syphilis does not leave behind any syphilitic changes in the milk teeth is not correct.

THE COMPLETION OF INADEQUATE TREATMENT OF PNEUMOTHORAX IN TUBERCULOUS CHILDREN BY ENDOPLEURAL INTERFERENCE. (THORASCOPY AND THE DIVISION OF ADHESIONS BY BURNING.) G. MEYERSTEIN, Ztschr. f. Kinderh. **43**:483 (May) 1927.

Cases are described which show the applicability of the method of Jakobaeus (the galvanic burning of pleural adhesions by direct vision) even in children. There is a possibility of benefiting, with its help, an inadequately treated pneumothorax so much that healing results, measured by the standard of continuous examination by the roentgen ray and the cessation of the excretion of sputum and bacilli, the temperature curve and the sedimentation reaction of the red blood corpuscles. Just how necessary it is to perform the operation cannot be decided with certainty in the early stages of pneumothorax therapy. That adhesions yield during the course of treatment, permitting adequate collapse without operative intervention, is known. One must study in each case a long time before deciding on galvanic division of adhesions, but in the event of failure, after perhaps several months, galvanic cauterization of the adhesions with the assistance of the thorascope is then indicated. In suitable cases, this represents a time sparing and fundamental influence on the pulmonary process. As in every form of pneumotherapy, a final decision as to the ultimate results of this treatment can naturally be given only after conclusion of the treatment and the collapse of the lung.

EMERSON, Boston.

GRAVE TUBERCULOSIS IN CHILDREN AND THE RELATIONS BETWEEN TUBERCULOSIS IN CHILDREN AND THE LATER CHRONIC PULMONARY TUBERCULOSIS. R. ZIMMERMANN, Ztschr. f. Tuberk. **47**:353, 1927.

On the basis of observations on about 100 children with severe tuberculosis, the following conclusions are reached: School attendance is apt to have an unfavorable influence on tuberculous children. Acute infectious diseases hasten the unfavorable outcome in tuberculous children. In children and adolescents from tuberculous families, the rather benign cirrhotic types are more frequently observed. It appears that children from such families have, as a rule, a better prognosis than patients from families that are free from tuberculosis. The placental transmission of tuberculosis should again be investigated. The anatomic differences between the primary pulmonary focus and foci of reinfection are not cleancut. In a general way the primary focus is poorly vascularized, surrounded by a gray capsule, frequently calcified, and located in normal lung tissue. The focus of reinfection has a richly vascularized black capsule; it calcifies much more rarely and is found in atelectatic tissue. The first manifestations of pulmonary tuberculosis in childhood are predominantly of the type of infiltrations. The beginning propagation of tuberculosis in the lung occurs mainly by blood and lymph stream. Tuberculosis at school age shows not infrequently the typical apical-caudal progression and may be of the cirrhotic cavernous type. The differences between tuberculosis in second childhood and that of adult age are differences of quantity and not of quality. During the premenstrual years there is a definite tendency for exudative manifestations.

MAX PINNER.

## Internal Diseases

THE TREATMENT OF HYPERTENSION. JAMES P. O'HARE, Am. Heart J. **2**:510 (June) 1927.

Prophylactic treatment aims at the avoidance of the hypertensive state in children of the so-called "vascular families" by teaching them to avoid excessive mental and physical strain as well as obesity. The actual treatment of the hypertensive person, of necessity aims largely at the reduction of the excessive vasoconstriction. This may be accomplished by a careful curtailing of mental and physical effort by enforced periods of relaxation, by diet, by drugs and probably by the liver extract when it is perfected.

A STUDY OF THE COURSE OF RHEUMATIC HEART DISEASE. FREDERICK A. WILLIUS, Am. Heart J. **3**:139 (Dec.) 1927.

One hundred and sixty cases of rheumatic heart disease were studied in which the data was available from the time of the initial infection till death from heart disease. Most of the cases occurred in the fifth decade (29 per cent), and only 19 per cent beyond that age. In 64 per cent, the first attack occurred before the age of 20, and in 85 per cent before the age of 30. In 124 cases there was mitral involvement only, and the average age at death was 30 years. In twenty-one cases there was aortic involvement only, and death occurred on the average at 43 years. Fifteen patients had both aortic and mitral involvement, the average age at death being 32 years. Recurrent rheumatic infections were most prevalent in the first decade.

MANNING, Santa Barbara.

THE VALUE OF OXYGEN FOLLOWING BRONCHOSCOPY IN CHILDREN. H. J. MOERSCH and W. M. BOOTHBY, Arch. Otolaryng. **6**:542 (Dec.) 1927.

In view of the experience with three patients, it is believed that it would be well to place all children in the oxygen chamber immediately after bronchoscopy, in order to decrease the labored breathing, to reduce cyanosis, to prevent edema as much as possible, not only of the larynx but of the respiratory membrane, and to reduce the possibilities of the occurrence of bronchopneumonia by keeping the patient in the best possible condition to prevent its development. Oxygen should be used, especially if laryngeal edema is marked and if pneumonia has developed, as it puts the patient in a better condition to overcome injury. If an oxygen chamber is not available, oxygen may be given through a portable oxygen tent or through a catheter, although the latter methods are usually inefficient and hard to carry out if the child is restless.

SANFORD, Chicago.

CELIAC DISEASE COMPLICATED BY PURPURA. FLOYD R. SMITH, Boston M. & S. J. **197**:658 (Oct. 20) 1927.

A white girl, aged 22 months, had indigestion, loss of weight and a distended abdomen. She was born at full term (forceps delivery), and weighed 7½ pounds (3,401.9 Gm.); she was breast-fed for six weeks but lost 2 pounds (907.18 Gm.). She did well on modified milk, and at 12 months weighed 23 pounds (10.4 Kg.). Shortly afterward, she developed pneumonia, with an apparently uncomplicated recovery. Later, she began to lose weight rapidly; she did not vomit and had a distended abdomen, with moderate constipation and mild diarrhea alternating; seven months after the pneumonia, she weighed 15 pounds (6,803.8 Gm.). She was put on a diet consisting of ripe raw bananas and was given up to eleven bananas daily. This was supplemented by small amounts of meat, fish, eggs, peas and other foods of high protein content. She gained 5 pounds (2,267.96 Gm.) in the next four months. She then began to lose her appetite, and the stools again became typical of celiac disease.

After three or four weeks, subcutaneous hemorrhages appeared, especially in the regions of the knees and elbows and over the thoracic vertebra; traces of blood were found in the urine and stools; the white cell count was 15,000, hemoglobin, 70 per cent, and coagulation time four minutes, but oozing from a puncture in the lobe of the ear continued for twenty-four hours. Extreme emaciation, sunken eyes and large abdomen were present. She was given 150 cc. of citrated whole blood intraperitoneally from the father, and 50 cc. of physiologic sodium chloride solution. There was no change in the condition for five or six days, when new hemorrhagic spots ceased to appear and the old ones vanished; the appetite returned and, except for occasional minor setbacks, improvement continued up to one year after treatment with bananas was started. The present weight is 27 pounds (12.2 Kg.), and the child appears to be unusually well. She takes five bananas daily, with a well balanced ration of other foods. Practically no milk has been tolerated since the treatment was undertaken.

GENGENBACH, Denver.

A STUDY OF THE INORGANIC CONSTITUENTS OF THE SERUM IN CHILDREN WITH ACUTE NEPHRITIS. KENNETH D. BLACKFAN and BENGT HAMILTON, Bull. Johns Hopkins Hosp. **41**:322, 1927.

Using the Fiske method for the determination of fixed base in the serum, the authors compare their observations in normal children and in children with acute glomerular and acute tubular nephritis.

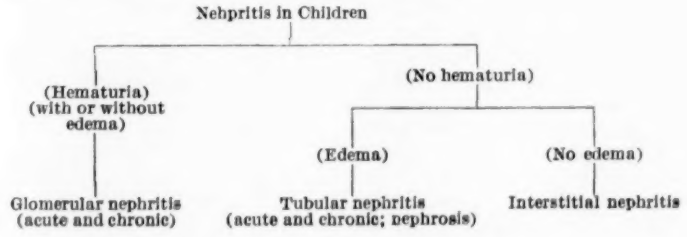
The total fixed base in normal children is constant between 156 and 161 cc. 0.1 normal base in 100 cc. of serum.

In tubular nephritis, most of the patients show a decrease of the fixed base in the serum and, in the acid radicles, a corresponding decrease of bicarbonate and undetermined acids. It is not known which factor is primary and which secondary. The authors think that the loss of protein through the kidneys is the cause of the low serum protein and that the albuminuria is the primary change.

In glomerular nephritis, there is a tendency to changes of the same kind that take place in tubular nephritis, but not so marked. It is noteworthy that in a few patients with alarming symptoms, the observations were quite normal.

TUBULAR NEPHRITIS (NEPHROSIS) IN CHILDREN AND ITS RELATION TO OTHER FORMS OF NEPHRITIS. WILBURT C. DAVISON and ROBERT SALINGER, Bull. Johns Hopkins Hosp. **41**:329, 1927.

The following schematic representation of nephritis is given. It is emphasized that mixed forms occur as the disease progresses.




There is a complete discussion of the clinical and laboratory manifestations in tubular nephritis and the results of various forms of treatment.

The authors summarize this work as follows:

"During the past fifteen years, among 53,965 ill children in the Harriet Lane Home, twenty-six have been diagnosed as having tubular nephritis (nephrosis). Six of the twenty patients regarding whom we have definite information are

well, and five of these have been so for more than seven years. Six of the twenty are dead. In eight, the disease is still active, although it cannot be said that they may not eventually recover. Unless our experience during the last ten years has been unique, it would seem that at least a fair proportion of children with tubular nephritis recover. However, very few reports of complete recovery are to be found in the literature and until the last few years there was no indication that the condition had been recognized by pediatricians. We cannot ascribe the recovery to any particular form of treatment; improvement and relapses may occur without any change in therapy. These cases indicate (1) that tubular nephritis (nephrosis) is a clinical as well as a pathological entity, which can usually be recognized by its clinical course; (2) that the etiology is as yet unknown; (3) that the prognosis is better than other reports would seem to indicate, although it must be emphasized that the duration may be long and that several years may elapse before the patient may be permanently free from edema and albuminuria, and (4) that at autopsy the tubules of the kidney are more markedly implicated than the glomeruli and interstitial tissue, although they, too, are damaged. We believe that the term, *tubular nephritis*, is preferable to *nephrosis* and that the presence or absence of hematuria and edema is the most satisfactory criterion at present for the classification of nephritis in children into its three principal anatomical varieties—glomerular, tubular and interstitial."

LYTTLE, New York.

STATUS THYMICOLYMPHATICUS CONSIDERED IN THE LIGHT OF RECENT WORK ON THE THYMUS. MAJOR GREENWOOD and HILDA M. WOODS, J. Hyg. **26**:305, 1927.

The concept of a status thymicolymphaticus so far as it is scientific belongs at present wholly to the statistical order of ideas and is in pari materia with the concept of racial susceptibility to disease. It is possible, for instance, that Swedish people are more susceptible to pulmonary tuberculosis than English. A practitioner confronted with a case of pulmonary tuberculosis might reasonably take the racial factor into account, and, other things equal, give a more cautious prognosis when the patient is Swedish than when he is English. But if the case had ended fatally much sooner than his general experience of the physical signs and symptoms of phthisis had led him to expect, he would hardly record on the death certificates: phthisis—Swedish nationality. The present use in certification and in evidence in coroners' courts of the phrases status lymphaticus and status thymicolymphaticus is, we suggest, a good example of the growth of medical mythology. A nucleus of truth is buried beneath a pile of intellectual rubbish, conjecture, bad observations and rash generalization. This heap of rubbish is described in the current scientific jargon and treated as an orthodox shrine. Indeed, when men of science speculate as to the nature of the antiepinephrine produced to excess in status thymicolymphaticus, one fells how well merited was the rebuke administered to some clinical endocrinologists by G. N. Stewart, who, in discussing certain papers on "hypoadrenal" constitutions, said: "In reading this paper and many others by 'clinical endocrinologists,' especially the French and the Italians, the physiologist can scarcely escape the feeling that here he has broken through into an uncanny fourth dimension of medicine, where the familiar canons and methods of scientific criticism are becoming foolishness, where fact and hypothesis are habitually confounded and nothing is but what is not." At the present moment we have not sufficient knowledge of the frequency of the single and combined anomalies which have been thought to define anatomically status lymphaticus or status thymicolymphaticus in a healthy population to permit us to assign the least importance to the recognition of these stigmas in the bodies of those whose deaths, apart from status lymphaticus, would in more pious, but not more superstitious, days have been attributed to the visitation of God.

TISSUE HYDRATION AND ITS RELATION TO SUSCEPTIBILITY AND IMMUNITY: AS SHOWN BY SKIN TESTS IN ASTHMA, CHRONIC SINUSITIS AND OTHER INFECTIONS. WILLIAM B. WHERRY, J. Infect. Dis. **41**:177, 1927.

In addition to well recognized qualifications parasites must be capable of freeing water in the tissues. It is suggested that interaction between the parasite and the host leads to the production of amines in susceptible persons and that the resulting hydration of the tissues favors the growth of the parasite.

Such interaction occurs when heat-killed bacteria are injected intradermally into human beings. The response varies with the species of bacteria and with the person. The production of local urticaria or this followed by immediate congestion is taken to indicate susceptibility; when the whole process is greatly aggravated the next day, the reaction is taken to indicate sensitivity. Antigens so selected and given by the desensitization method yield excellent therapeutic results due to the production of opsonins and of a lessened tendency to tissue hydration.

AUTHOR'S SUMMARY.

ASTHMA IN CHILDREN. VII. COMPARATIVE METHODS OF SKIN TESTING WITH DIFFERENTLY PREPARED EXTRACTS OF HOUSE DUST. M. MURRAY PESHKIN, J. Lab. & Clin. Med. **13**:67 (Oct.) 1927.

Fourteen asthmatic children were tested by scratch and intradermal methods with concentrated fluid extract of house dust (Coca), dry powdered house dust, and a specially prepared solution of powdered house dust. The intradermal method was found superior to the scratch, and the solution of powdered house dust from ten to one hundred times more potent than the concentrated fluid extract (Coca).

THE EFFECTS OF THE INTRACUTANEOUS INJECTION OF EPINEPHRINE IN CHILDREN. J. D. PILCHER, J. Lab. & Clin. Med. **13**:201 (Dec.) 1927.

Pilcher injected children from a few months to 12 years of age, intracutaneously, subcutaneously, and intramuscularly with 0.2 mg. of epinephrine. The resultant rise in blood pressure reached its maximum more rapidly, and attained a higher maximum by the intracutaneous route than by the other two methods. The intramuscular route closely approached the intracutaneous in efficiency.

GIBSON, Elkhart, Ind.

FRAGILITAS OSSIUM. ITS HEREDITARY ASPECT WITH CASE REPORT. M. S. ROSENBLATT, J. Michigan M. Soc. **26**:292, 1927.

The patient, an American girl, aged 12, had a fracture of the femur and had previously had fifteen fractures of the long bones. From her maternal great-grandfather had descended nine sons and (from one son) eight grandchildren (four boys, four girls) all of whom had multiple fractures. From the histories, apparently only one of these (a grandchild, the mother of the patient) had blue sclerae. The expectation that only 50 per cent of the offspring of an affected person married to an unaffected person will be affected did not hold true in this family, and neither did the rule (Key) that in such families the offspring with normal sclerae will have normal children. Attention is called to the differentiation of osteopsathyrosis idiopathica, osteogenesis imperfecta and hereditary hypoplasia of the mesenchyme which are mentioned as synonyms by Osler.

AUTHOR'S SUMMARY.

HYPERTROPHIC CIRRHOSIS OF THE LIVER IN INFANCY AND CHILDHOOD. EARL MENDUM TARR, M. J. & Record **126**:293 (Sept. 7) 1927.

Cirrhosis of the liver is relatively rare in infancy as is also disease of the gallbladder. Contrary to the views of many observers, both these condi-

tions are found not infrequently in children, aged from 3 to 12 years. The livers of infants and young children are susceptible to injury and any form of injury to hepatic tissue in them must be looked on with grave concern. The disease occurs more often in boys. One of the chief causes of the condition is that of toxicity, and acute infectious fevers have been known to excite the disease. One of the first and most constant symptom is some form of digestive disturbance.

Loss of weight and loss of appetite are observed. The skin is sallow, and later jaundice is present. Careful palpation reveals the evenly enlarged liver. The blood shows a secondary anemia. McClure, Mendenhall and Huntsinger proved by certain studies that magnesium sulphate exhibits a profound effect on the functional activity of the liver. It was their contention that this drug would favorably modify the function of the liver deranged by disease. The action of the drug depends on the amount which reaches the liver. The author then gives reports of cases in which magnesium sulphate was given. He gave 20 cc. of a 2 per cent solution intraperitoneally twice weekly. Keen observation on the part of those in general practice who see most of these patients should be encouraged and reports of such cases, for obvious reasons, should be published.

SOME POINTS IN THE DIAGNOSIS AND TREATMENT OF CARDIAC CONDITIONS IN CHILDREN. T. EPSTEIN, M. J. & Record **126**:298 (Sept. 7) 1927.

Because of their unmitigated condition, their permanent disability, and their slow recovery, the large number of children with heart disease have become one of the most important of sociologic problems. In the majority of these cases there is a general rheumatic infection which has selected the heart for its greatest pahtologic attack. Children with heart disease usually die from the disease. Adults with heart disease die from septicemia, uremia, embolic infarctions, cerebral hemorrhage or coronary thrombosis. Heart disease in infancy is usually congenital. In the first two or three years of life, the disease is mainly due to septic infections and after the third year is chiefly caused by rheumatism. The means by which the various kinds of heart disease may be diagnosed are mentioned. Every child with rheumatic diathesis should be under observation. The cardiac clinic should include the precardiac child. Special care should be given to the heart in every illness which increases its rate or disturbs its rhythm. Complete physical and mental rest will relieve the heart of a great deal of its extra work. The treatment of acute, subacute and chronic endocarditis as well as congenital heart disease is then discussed.

The general plan of treatment of the cardiac child is the prevention of any infectious disease, of rheumatism, the radical treatment for any heart damaging illness and the careful management of the already damaged heart. In endocarditis, the child should be given salicylates for two weeks. Guaiacol carbonate with quinine sulphate was given for the next two weeks. Tonic treatment for the following two weeks should be prescribed. This therapeutic cycle may be repeated at biweekly intervals over a long period of time. The prognosis of heart disease in children in the majority of cases is doubtful and discouraging.

THE MECHANISM OF DEATH IN STATUS LYMPHATICUS. EDWARD A. TRACY, M. J. & Record **126**:302 (Sept. 7) 1927.

Paultauf showed that a condition of constitutional inferiority manifested by a pronounced lymphatism produced a liability to sudden death, the death being due to heart failure.

Wiesel showed that status thymicolymphaticus was always associated with a hypoplasia of the chromaffin tissue. He believed that sudden death was due to lack of epinephrine and was caused by a sudden increase in vagal tonus. Hormones in the blood stream are acted on by stimuli passing to them from appropriate nerve fibrils, and they in turn act on the musculature of the blood

vessels. These hormones are classified according to their action, as diastolic or systolic. The diastolic produce dilatation and the systolic, contraction of the musculature; epinephrine and pituitary are systolic hormones.

The thyroid and thymus glands may be sources of diastolic hormones. These hormones activate both the musculature of the heart and the blood vessels of the skin. Stimuli passing over the vagus fibers to the heart cause it in the presence of diastolic hormones to go into diastole, and stimuli passing over the sympathetic fibers cause it in the presence of systolic hormones to go into systole.

Without these hormones, there cannot be diastole or systole of the heart. That death in status lymphaticus is caused by lack of epinephrine is correct. In that condition the diastolic hormone is in excess, the systolic insufficient, so that the heart is found invariably in diastole.

HYPERTROPHIC PYLORIC STENOSIS. SAMUEL A. BLAUNER, M. J. & Record **127**:304 (Sept. 7) 1927.

Since Armstrong, in 1771, and later Beardsley, in 1777, called attention to this disease, physicians can readily recognize the condition, but there has not been unanimity of opinion regarding its etiology and pathology, and the treatment is solely a matter of choice, according to one's own experience with the disease. During the last decade surgical treatment is in the ascendency, at least in America. The Germans, who in general are still opposed to operation, obtain as satisfactory results by medical treatment as are obtained at surgical clinics. The pathologic changes of the disease are fully discussed. In an interesting report of twenty-three autopsies on patients who had undergone Ramstedt's operation at the Baby's Hospital, but who had died later from some other cause, it was stated that all showed a normal pylorus. The incision should not affect the hypertrophy; it should only release the contraction due to spasm. The symptoms of the disease are enumerated. Roentgenographic and fluoroscopic examination should be a great diagnostic aid and should, in a way, be confirmatory of the other symptoms present.

In treatment, one's aim should be to eliminate as far as possible the element of risk and to apply the method best adapted to the case. Most cases show sufficient degree of spasm to justify medical treatment at the beginning. Progress of the case is best shown by the degree of emaciation. In medical treatment, use of atropine, if properly carried out, is the method of choice.

WILLIAMSON, New Orleans.

DIABETES IN CHILDREN. E. S. WEGNER, Nebraska M. J. **8**:417, 1927.

A series of cases of patients with juvenile diabetes is reported. The urine should be kept sugar free and infections avoided to prevent "strain" and increased damage to the pancreas.

HAMILTON, Omaha.

COURSE AND PROGNOSIS OF DIABETES MELLITUS IN CHILDREN. GLADYS L. BOYD, Canad. M. A. J. **17**:1167, 1927.

This paper is based on the study of ninety-five patients with diabetes mellitus under 15 years of age.

Fifteen per cent of these children were able to take an adequate diet without insulin, but in none of them did the initial tolerance exceed 100 Gm. of carbohydrate daily. Three fourths of the remaining 85 per cent could not be kept aglycosuric on their basal diets without insulin. Thirty-eight per cent had coma. In the series studied, fourteen children have already lived for from four to four and one-half years and twenty-nine have outlived the two and one-half years previously allotted to them by Joslin. In sixteen of twenty given insulin, normal weight was attained at the end of the first year and maintained at or about that level for the three year period since. Gains in height accompanied the gain in weight.

There was a steady, definite improvement of tolerance in nine out of the thirty-seven cases studied, as shown by the decreased requirement of insulin.

The author also states that the evidence of regeneration, as noted in the pancreas after death from some other cause in patients treated with insulin, shows that this process actually seems to occur.

The incidence of diabetic coma has not declined, but a material decrease in its mortality has occurred, due to insulin and to earlier recognition and treatment.

MOORE, Omaha.

DIAGNOSIS OF BRONCHIECTASIS AND LUNG CAVITIES BY INTRATRACHEAL INJECTIONS OF LIPIODOL. P. F. ARMAND-DELILLE, Tubercle **8**:557 (Sept.) 1927.

As a general rule this method should be employed in all cases in which the stethoscope reveals the symptoms of cavity, when one cannot say with certainty whether a cavity is really present, or when one cannot determine the nature or origin of the cavity. In cases of vomiting, it may be of great service in determining whether the pus comes from above or below the diaphragm.

After a description of the technic of the injection, the author describes the various forms of bronchiectasis which may be revealed by this method. Ampular bronchiectasis is the most characteristic and most important variety, and causes the most marked general and local symptoms. Glove-finger dilatation is rarer and occurs in patients with symptoms of chronic bronchitis, but without signs of cavity. It is characterized by large dilatations in the lower part of one or both lungs. The lowest bronchi are irregularly enlarged and end in a culdesac like the finger of a glove. In a third type there are regular, cylindric dilatations of the bronchi in the region of the hilum. A whole or a part of a bronchus is regularly enlarged without lateral dilatation. A fourth type showing small fusiform dilatations of the small bronchi is most frequently seen in children who have suffered from whooping cough with bronchopneumonia. Irregular cylindric dilatations may occur in children with sclerosis of the lungs. In these cases, the deformity is probably due to traction on the bronchi by the sclerosed lung.

In the author's experience the procedure is free from danger, and in many cases of bronchiectasis seems to be of distinct therapeutic as well as of diagnostic value.

OLEOTHORAX. F. G. CHANDLER and S. ROODHOUSE GLOYNE. Tubercle **8**:563 (Sept.) 1927.

The following indications for the production of an oleothorax are suggested:

To replace a pleural effusion when it is desired to maintain collapse of the lungs and to prevent the development of an obliterating pleurisy.

As a method of producing collapse of the lungs when frequent injections of air are undesirable or impossible, or when permanent collapse is indicated, as in basal bronchiectasis.

To relieve the pain or discomfort of too high negative pressure in the pleural cavity.

To replace pleural effusion rich in tubercle bacilli.

To replace tuberculous empyema.

To replace secondarily infected pleural effusion.

In cases of spontaneous pneumothorax with permanent pleuropulmonary fistula. The oil may close the mouth of the fistula and promote its healing.

In other types of pleuropulmonary fistula, as after an empyema is coughed up.

Possibly in the case of a chronic empyema with thickened pleura and failure of expansion of the lungs when decortication is not advisable. Possibly as a means of preventing firm adhesions.

The oils used are sterilized olive oil, liquid paraffin and oil of gomenol, from 5 to 20 per cent.

From their clinical and experimental work, the authors conclude that:

When used to replace pleural effusion in man, gomenol appears to be harmless, and in certain cases is of great clinical value.

Experimentally, iodized oil 40 per cent and gomenol do not have any effect on the pleural membrane of normal rabbits beyond the production of a few soft, sterile adhesions.

In experimentally infected rabbits, the adhesions produced by gomenol show histologic signs of tuberculosis.

There is no evidence of any lipolytic action when oils are injected into the pleural cavity.

Neither of these oils has any toxic effect on distant tissues.

The bactericidal effect of iodized oil and gomenol on *B. tuberculosis* in vitro is not great.

Paraffin wax injected into the pleural cavity of rabbits remained sterile for many weeks. However, firm adhesions are produced all around it.

SMITH, Ogden, Utah.

MYELOMA OF HUMERUS. E. A. BROOK, Proc. Roy. Soc. Med. **20**:62 (Aug.) 1927.

Brook reported a case in a boy, aged 13 years. The patient received an impacted fracture of the surgical neck of the right humerus in August, 1925, which healed with sound union and full restoration of function. In May, 1927, he fractured the upper third of the right humerus. The roentgenogram showed a spontaneous fracture through a myeloma. The roentgenogram made two years previous did not show evidence of myeloma. The point of interest was the question of relationship between the original injury and the present condition: Was there such a relationship or was it a pure coincidence?

DIAPHYSICAL ACLASIA. E. A. BROOK, Proc. Roy. Soc. Med. **20**:69 (Aug.) 1927.

This condition was found in a child, aged 9 years, who was admitted to the hospital with subacute appendicitis. Roentgen-ray examination showed that every long bone in the body had irregular growths close to the epiphyseal line. The patient also had marked genu valgum and also showed evidence of past rickets which, apparently, was a factor in the production of the multiple exostoses.

A CASE OF GANGRENE OF THE LEG AND ARM. WALTER BROADBENT, Proc. Roy. Soc. Med. **20**:71 (Oct.) 1927.

A boy, aged 2½ years, was admitted to the hospital complaining of pain in the right leg when he ran about. The next morning his eye and leg were black. Two days later, the left leg began to become black, and there was well-marked hemorrhage under the skin of the left forearm and hand. The temperature remained at about 99 F. for the first week. Later, when the gangrene was progressing, it became hectic, rising to 101 or 102. The condition of the face cleared up in about three weeks. The limbs were kept dry in cotton wool and allowed to separate without any surgical intervention. The hand and wrist came off in about seven weeks and the foot and a portion of the leg somewhat later.

The author was unable to offer any explanation of the cause of the original illness.

A CASE OF PERFORATION OF THE BOWEL IN A BABY. HIRSCHPRING'S DISEASE. J. R. GRIFFITH, Proc. Roy. Soc. Med. **20**:74 (Oct.) 1927.

An infant, aged 6 months, was admitted to the hospital suffering from severe abdominal pain and tenesmus; on examination, a sausage-shaped lump was felt in the pelvis. A diagnosis of intussusception was therefore made and the

abdomen opened. A section of the pelvic colon was found to be edematous, thickened and immobile, and pitted on pressure. The bowel was perforated lower down. A tube was tied into the bowel and a colostomy performed. The child unexpectedly recovered.

WILLIAMSON, New Orleans.

BRONCHITIS IN INFANTS. P. LEREBOULLET and F. SAINT GIRONS, Paris méd. **46**:394 (Nov. 12) 1927.

Bronchitis in the first year of life is of such frequent occurrence that methods of combating it should be definitely undertaken. Hygiene should first be considered. The child should be isolated, and the air kept warm and moist, at about 18 C. Eucalyptus oil in steam is helpful. Skin friction over the child's chest promotes good circulation, and this can be done if the chest is rubbed with some ointment. Olive oil and essence of turpentine are as satisfactory as any other.

Secondly, as rhinitis is always the beginning of the condition, the nose should be disinfected with a silver preparation. Hydrotherapy will take care of the temperature. This should be a bath of from 37 to 38 C. for from five to ten minutes.

Lastly, the authors believe that in severe cases hypodermic injections of camphor in oil are helpful.

SANFORD, Chicago.

PROGRESSIVE MYOSITIS OSSIFICANS. G. BERGHINZ, Clin. Pediat. **9**:103, 1927.

A case of myositis ossificans progressiva in a girl, aged 6 years, is reported. There was no history of syphilis, and the Wassermann reaction was negative. Few possible etiologic factors are given. The supposition that the condition was a constitutional anomaly was supported by the fact that at birth morphologic changes of the hands and feet were observed. The possibility of a trophoneurotic factor was indicated by the symmetrical arrangement of the ossifications. The therapeutic measures used were all ineffectual, and the course of the disease is one of gradual progression.

ESSENTIAL MICROSOMIA. V. DEBENEDETTI, Clin. Pediat. **9**:421 (May) 1927.

The author presents a case of microsomia in a boy, aged 9 years and 5 months, 87.5 cm. tall and weighing 10,600 Kg. At birth, the child was so small that his bed was made in an ordinary shoe box. His general health was always good, and his growth increased propertionately. There were no gross disproportions in the various parts of the body such as is often observed in persons with infantilism.

The author feels justified in using the term microsomia to denote only those persons whose development at birth was a miniature of the normal and whose future development follows a course proportionately the same as that of a normal person. Especially in later life, these persons show some signs of attenuated endocrine deficiencies. At birth, many of them show a partial or complete cryptorchidism.

SODOKU (RAT-BITE FEVER). G. TACCONE, Clin. Pediat. **9**:595 (Aug.) 1927.

Sodoku is the name given by the Japanese to a symptom complex observed in persons bitten by rats. After a long series of bacteriologic studies, Foutaki found a spirochete in the lymphatic glands, blood and skin of patients suffering from the disease. He called the organism *"Spirochaeta morsus muris."* Other observers called the same organism *"Spirochaeta japonica."* Two forms of spirochete have been described. The one found in the lymph glands is long and measures from 9 to 10 microns; the one found in the local lesions and the blood is shorter (from 2 to 6 microns) and has a flagellum at both extremities. The disease may be reproduced in various animals by inoculating

them with pathologic material containing the spirochetes but not with cultures. The disease is transmitted to man in the majority of cases through a rat bite or through animals who feed on rats (cats, ferrets, weasels).

The incubation period is from eight to twenty-five days. Inflammatory changes are first observed around the area of the bite. The tissues become hard and present a violet hue. A concomitant lymphangitis occurs with a regional adenopathy. If secondary infection sets in, gangrene and edema may follow. Fever is a constant symptom and may be accompanied by all or any of the following symptoms: chills, general malaise, headache, insomnia, anorexia, melancholia, delirium, muscular cramps and pains in the bones or joints. The temperature curve is somewhat irregular and drops by crisis with profuse sweating. An exanthem characterized by a polymorphous eruption may occur early or late in the disease. It is generalized and consists of well delineated patches. It is usually red or of a reddish-violet tint. The cutaneous lesions show a lymphocytic infiltration with many plasma cells and epithelioid cells in the derma. In the lower layers there is a nodular infiltration limited for the most part to the lobules of the fatty tissue.

The following clinical varieties of the disease have been described: (1) Cases in which local symptoms prevail (infection, gangrene, edema); (2) the febrile form with a generalized exanthem; (3) the painful form, differentiated from the preceding by the predominance of pain as a symptom of onset; (4) the nervous form with disturbed sensation and motor symptoms (paresthesias, paralyses, diminished reflexes and exanthem). The following complications have been observed: retro-ocular cellulitis, icterus, nephritis, pleuritis, phlegmon of the foot, iritis with the formation of tuberculoid nodules, corneal ulcers, albuminuria, cardiac arrhythmia and laryngitis. Splenomegaly and hepatomegaly may also occur.

In Europe, the prognosis is good. In Japan, before the introduction of arsenical therapy, the mortality was about 10 per cent. Mercury and bismuth have been used in the treatment of sodoku with indifferent results. Most authors are agreed that arsphenamine therapy is the most efficacious. The author reports a typical case in a child, aged 2 years. After fifteen days the child presented a generalized erythema with marked tracheobronchial adenitis. The diagnosis was made by finding the spirochetes in the urine and by a positive Wassermann reaction with the blood. Arsphenamine therapy was instituted with good result.

ESOPHAGEAL STENOSIS AND CARDIOSPASM. G. CAREDDU, Clin. Pediat. **9**:641 (Aug.) 1927.

The author reports a case of esophageal stenosis in an infant, aged 28 months, and a case of cardiospasm in a child, aged 3 years. In the first case, the stenosis was due to an anatomic defect of congenital origin, and the cardiospasm was probably due to a vagotonia with perhaps a latent tetany.

The patient with the stenosis was considerably improved by the administration of antispasmodics, ultraviolet therapy and the systematic dilatation of the esophagus with sounds. The patient with the cardiospasm was cured following treatment with ultraviolet rays.

SYNTHALIN IN DIABETES MELLITUS. G. TACCONE, Clin. Pediat. **9**:753 (Oct.) 1927.

Taccone reports his impressions following the use of synthalin, a guanidine derivative, in children suffering from diabetes mellitus. Synthalin may be given by mouth, and its action is slower but more prolonged than that of insulin. It is valuable in cases in which the use of insulin is refractory. A combination treatment with insulin and synthalin reduces the number of injections necessary in using insulin alone. By its use, the patients are allowed much more freedom in their diet than with insulin. It diminishes the appetite

and by so doing offers a means of controlling diabetic patients with voracious appetites. Synthalin also possesses many disadvantages. It is very toxic and is not tolerated by many patients. Even the smallest therapeutic doses frequently produce a marked intolerance. It is of little value when prompt action is required, as in precoma and coma. In children, it is of even less value than in adults. In the author's experience, children are very intolerant to it. The patients treated with synthalin, although sugar-free, fail to have the feeling of well-being and the general improvement noted with the use of insulin. Modifications in its chemical structure are necessary before it can be considered as a substitute for insulin. ZAMBARANO, Northville, Mich.

ENDOCARDITIS LENTA IN CHILDHOOD. E. NEDELMANN, Arch. f. Kinderh. **81**:106 (June) 1927.

Endocarditis due to *Streptococcus viridans,* although relatively frequent in adult life, is rarely seen during childhood. The clinical picture is typical, namely, an insidious onset frequently with malaise and rheumatoid pains, a palpable and often tender spleen with irregular or intermittent fever which may last for months, and secondary anemia. Heart murmurs may be entirely absent in the early stages of the disease, except when the infection is superimposed on an old rheumatic valvular lesion. Almost pathognomonic is the occurrence of multiple small emboli, which may cause hemorrhages in the skin, or painful lesions simulating an early furuncle, panaritium, or even erysipelas, but never progressing to formation of pus. Emboli may also lodge in various internal organs. The diagnosis is verified by obtaining *Streptococcus viridans* from the blood stream; once this is established, the prognosis is unfavorable.

A typical case, occurring in a boy, aged 13, is described, confirmed by the clinical course, blood culture and autopsy. HEMPELMANN, St. Louis.

JUVENILE HYPERTROPHIC PERIOSTITIS. H. HOLTHUSEN, Beitr. z. path. Anat. u. z. allg. Path. **77**:318, 1927.

Holthusen describes differences, as determined roentgenologically, in the hypertrophic periostitis or hypertrophic pneumic osteo-arthropathy of Bamberger-Marie of young growing children as compared with adults. In the latter the periosteal thickening predominates over bone formation, whereas in juveniles, in whom bone formation is still active, a greater degree of periosteal bone formation occurs. Holthusen's patient was a girl, aged 18, with marked evidences of infantilism.

CROSSED DYSTOPIA OF THE THYMUS. P. SCHNEIDER, Beitr. z. path. u. z. allg. Path. **77**:449, 1927.

Developmental anomalies of the thymus are rare. Schneider briefly reviews the previously recorded examples and describes, as a condition not previously noted, crossed dystopia of the thymus in a boy, aged 11. From the normally situated upper cervical portions the larger lower portions of the two lobes crossed each other, the main portion of the left lobe lying anteriorly and on the right side, and the right lobe on the left side. O. T. SCHULTZ.

RUPTURE OF A DIAPHRAGMATIC HERNIAL SAC. J. LÜNING, Centralbl. f. allg. Path. u. path. Anat. **40**:97, 1927.

An apparently normal boy, aged 3 days, died suddenly after marked dyspnea. At the autopsy the diaphragm in the left tendinous part contained a smooth-shaped opening 3.5 by 2.5 cm., through which a sac the size of a goose egg protruded that was lined with peritoneum and filled with everything in the peritoneal cavity except the liver, 5 cm. of ileum, the first part of the ascend-

ing colon and the rectum. Two tongue-like projections, the largest 25 by 20 by 4 mm., extended from the left lobe of the liver into the sac. A recent tear in the sac 5 cm. long with blood-stained edges permitted several loops of small bowel to enter the left pleural cavity. The left lung was atelectatic, and the mediastinum displaced to the right of the midline. Presumably the hernial sac burst after the stomach was distended with food.

GEORGE RUKSTINAT.

RECURRENT SWELLING OF THE PAROTID. S. WOLFF, Jahrb. f. Kinderh. **112**:334 (May) 1926.

The infant brought to Wolff's clinic had intestinal spasms and typical symptoms of ileus. Later, spasms of the esophagus and severe pyloric spasms appeared. While the duodenal sound was being passed, a marked swelling of both parotids occurred, with the superior lobe in relief as in typical mumps. The submaxillary gland was enlarged on both sides. The parotids were stony in consistence. After two hours, the symptoms disappeared without any treatment. This occurrence repeated itself two months later and four months later. After eliminating other possible explanations, the author states his belief that there was a spasm of Stenson's duct, on account of which the discharge of saliva was checked. The stony swellings are to be explained by salivary stasis.

CHRONIC PERITONITIS IN CHILDHOOD. ERICH ROMINGER, Jahrb. f. Kinderh. **113**:1 (June) 1926.

In four cases reported by Rominger, there was pot-belly, a final result of a chronic peritonitis not caused by tuberculosis. In three cases, biopsy demonstrated the absence of tuberculosis. There was no tendency to adhesions. Old tuberculin did not cause any reaction. The children had good appetites, and fever was not present. In the first case, the pot-belly had been present for six years.

BIERMER'S ANEMIA IN CHILDHOOD. MAX BRÜCKNER, Jahrb. f. Kinderh. **114**:370 (Dec.) 1926.

Brückner reports two cases of Biermer's anemia in girls aged 10 and 12 years, respectively. In the first case, megaloblasts were absent in the blood, at least in the last weeks of life. The clinical diagnosis of Biermer's pernicious anemia was confirmed at necropsy. In the second case, a probable diagnosis of endocarditis was made during life. Macroscopic manifestations at necropsy indicated pernicious anemia. Microscopic examination was not made. Not only in early childhood, but in school years the blood picture of pernicious anemia is not of such great diagnostic importance, as might appear from the textbook, since the diagnosis can be made with certainty only from the clinical course. Retinal hemorrhages as seen in the first patient are also observed in anemias secondary to carcinoma, but these would scarcely appear in childhood.

PACE, Chicago.

BLOOD TRANSFUSIONS IN SEVERE PYURIA. LUDWIG SILBER, Monatschr. f. Kinderh. **36**:499, 1927.

In three cases of severe pyuria, the author obtained good results with from five to six intramuscular injections of from 10 to 20 cc. of blood.

ACUTE LIVER INSUFFICIENCY IN CHILDHOOD. HERMAN LENHARTZ, Monatschr. f. Kinderh. **37**:139, 1927.

In the course of an epidemic of catarrhal icterus with gastro-intestinal symptoms, a boy, aged 4 years, developed what appeared to be the same disease. He grew gradually worse, and finally coma developed. Death occurred in

convulsions. The examination of the liver did not disclose the usual necroses typical of atrophy. If the condition were the result of ordinary catarrhal jaundice, that name is a misnomer. Jaundice is purely a symptom. Hepatitis would be a better word. Possibly all cases of catarrhal jaundice show such a pathologic condition. The author believes the term "liver insufficiency" should be applied to these conditions.

AUTOTHERAPY OF EMPYEMA IN INFANTS. FLESCH, Monatschr. f. Kinderh. **37**:149, 1927.

Empyema in infants has a notoriously bad prognosis. A number of authors have recommended aspiration of the fluid and the reinjection of from 0.5 to 1 cc. of the fluid beneath the skin. The author reports two cases in which this treatment gave excellent results.

GERSTLEY, Chicago.

DIABETES IN CHILDREN. RICHARD WAGNER, Wien. klin. Wchnschr., Separate Pamphlet, Oct. 13, 1927, Vol. 40.

Wagner presents a thorough resumé of his experiences in treating diabetic children since the introduction of insulin. Approximately fifty children are now under his care in the diabetic service of the Wiener Universitäts Kinderklinik. Since insulin was introduced, death has not occurred in any of the children whose parents adhered to instructions.

In regard to etiology, the author leans toward the view that juvenile diabetes is an hereditary, endogenous degeneration. This is supported by Wieland's recent demonstration of Mendel's law as applied to diabetic families. Wagner observed that, in the hereditary transmission of diabetes, the age of incidence becomes progressively lower. He holds that infections and excessive carbohydrate diets are not direct causes; they merely break down a previously defective islet system.

For the most part, the author employs a permanent diet containing 10 per cent protein and carbohydrate equivalent to 100 Gm. of white bread. The remainder of the required calories is supplied by fat. He regards this as a middle ground diet which is adaptable to home use. Since the report by Adlersberg and Porges of improvement in tolerance following use of a high carbohydrate diet and increased doses of insulin, the author has in part confirmed their experiments in juvenile diabetic patients. The diet used contained 36 per cent carbohydrate. He has found, however, that this diet results in a marked tendency toward hypoglycemia, which renders it objectionable for treatment at home. Together with MacGillivray, he studied the blood sugar tolerance to fixed doses of carbohydrate and insulin in children who were receiving the types of diet already described. In the diabetic patients on the high carbohydrate diet, there was a greater drop and a more rapid return to normal than in those on the permanent medium carbohydrate diet. Apparently a liver rich in carbohydrate is more unstable with respect to metabolism of glucose than is one filled with fat. The author regards the principal value of the high carbohydrate diet to lie in its ability temporarily to provide a varied and palatable menu which will stimulate rapid growth. He does not feel convinced that it increases sugar tolerance.

Wagner makes frequent use of Falta's meal-fruit diet which is poor in protein, when there is a temporarily decreased tolerance due to infection or laxity in management at home. It is likewise indicated in patients who are resistant to insulin.

In a comparison of views on total caloric needs of juvenile diabetic patients, Wagner finds that American physicians have tended to keep the children below standard weight, while the European physicians have reported figures showing normal or overweight children. Since Joslin has observed constant or increasing tolerance in his young patients, the author concedes that a lean state of nutrition might be preferable. Normal progress in regard to increase and sexual development is of main importance.

The sugar content of vegetables, according to Wagner, is subject to wider variations than is stated in standard texts on diabetes. The method of cooking is also a determining factor. In conjunction with Warkany, he investigated, by means of blood sugar curves, the carbohydrate values of common vegetables in terms of the white bread equivalent. The table showing the results is included in his paper.

The author found that children differ markedly from adults in the glucose equivalent to a unit of insulin. His formula for this glucose equivalent is:

$$\text{Dextrose Equivalent} = \frac{100\%\ \text{C} + 58\%\ \text{P} + 10\%\ \text{F} - \text{Excreted C}}{\text{Units Insulin per 24 hours}}$$

This is not a constant figure. It may vary as much as 100 per cent, with changes in the relative proportions of the food constituents. It is smaller on a diet high in fat and greater on a diet rich in carbohydrate. It is altered during infections. Duration of the diabetic process is a factor: the longer the duration, the less the dextrose equivalent. In children, it decreases with advancing age.

According to the author, one must approach each case empirically in order to determine the required dosage of insulin. Tables showing dosage based on carbohydrate content of the diet are worthless.

The amounts Wagner employs range from 10 to 80 units. When two doses are given daily, the proportion is 6:4 and the interval twelve hours. When three doses are indicated, he proportions the doses according to the ratio 5:2:3, injecting the insulin at 5 a. m., 2 p. m. and 9 p. m. During the course of intercurrent infections he maintains or increases the dosage, and gives a low calorie diet on certain days or causes the patients to fast or receive only fruit. He employs lemonade in patients displaying anorexia. Patients with diarrhea receive daily one-half liter of milk and 32 Gm. of chocolate.

As to the future progress of juvenile diabetic patients, Wagner is optimistic. He has watched them grow normally and attain sexual maturity. They become good scholars and are able to participate in sports. They are more deserving of aid from the state than are the mentally deficient. He admits that, if the hereditary nature of diabetes is accepted, reproduction by diabetic persons for several generations will act harmfully for the race. OLMSTED, Seattle.

GENERALIZED INFANTILISM. P. SCHURMANN, Virchows Arch. f. path. Anat. **263**:649, 1927.

A woman, aged 25, was 143 cm. tall and weighed, before her final illness, 46.2 Kg. The psyche was infantile; she had never menstruated, and the secondary sexual characters were not developed. She died in diabetic coma. The chief manifestation at necropsy was absence of both ovaries, congenital in origin. This was held to have led in turn to the hypoplasia of the pineal, thyroid and pancreas, to the hypertrophy of the hypophysis, and to the generalized microsomia also noted at necropsy. O. T. SCHULTZ.

## Nervous Diseases

THE SO-CALLED SMALL ROUND CELL INFILTRATIONS: I. POLIO-ENCEPHALITIS AND ACUTE EPIDEMIC ENCEPHALITIS. N. A. MICHELS and J. H. GLOBUS, Arch. Path. **4**:692 (Nov.) 1927.

In a study of twelve cases, it was found that the vast majority of the infiltration elements in the adventitial spaces, as well as those in the extravascular territory in inflammatory lesions of the central nervous system, are cells that filtered out in some selective fashion through the endothelial walls of the local blood vessels. These cells tend to be extravasated in showers.

As the endothelium of the blood vessels never exhibited any cytogenetic activity, it was considered proof that the exudates were carried to the site of inflammation by way of the blood stream. After extravasation, the behavior of the lymphoid cells varies. Some remain as lymphocytes and large mononuclears; others undergo further differentiation and become transformed into polyblasts, macrophages, granular cells, and occasionally into plasma cells, the prevailing differentiation being polyblastic.

Extravasation of red blood corpuscles was noted in each case, and while it was generally restricted to the perivascular zone, in many cases it assumed hemorrhagic proportions. The blood stream breaks through a partially disintegrated endothelial lining of a vessel and flows freely into the general parenchyme. In cases of encephalitis which run an exceedingly stormy course with a sudden and abrupt termination, the predominant histologic picture is that of numerous hemorrhages and pronounced extravasations of red blood cells with an unusual sparsity of typical small round cell infiltrations.

SANFORD, Chicago.

THE ACID-BASE COMPOSITION OF URINE FROM AN EPILEPTIC CHILD. JAMES L. GAMBLE and BENGT HAMILTON, Bull. Johns Hopkins Hosp. **41**:389, 1927.

Gamble and Hamilton studied the acid-base factors in the urine of an epileptic girl, aged 11 years. The attacks lasted two days, with a free interval of about a week. Urine was obtained for twenty-nine days while the child was on a constant diet and fluid intake.

During periods of seizures, there was a greatly increased excretion of total acid, mostly chloride ion and of total fixed base, mostly sodium. Urinary acidity and ammonia remained nearly stationary. This is thought to indicate a loss of extracellular body fluid in the urine during periods of active epilepsy. In corroboration of this, corresponding increments of urine volume were found.

LYTTLE, New York.

CONGENITAL AMYOTONIA: REPORT OF TWO CASES. C. C. RUDOLPH, J. Florida M. A. **14**:234 (Nov.) 1927.

The disease is congenital, though in the majority of cases it is not recognized till some time after birth. The etiology is unknown, and sex does not play a part in the condition. The outstanding symptoms are flaccidity and loss of power of the muscles, which, in contradistinction to the Werdnig-Hoffman syndrome, do not show a tendency to progressive increase. No instance of complete recovery has been recorded, though in a number of instances decided improvement has been noted.

CASE 1.—An infant, aged 10 months, showed symptoms at birth; the family history and physical examination were negative except for signs of rickets at 10 months and marked flaccidity of the muscles, hypermobility of the joints, atonicity, which was most marked in the shoulder girdle, extremities and neck, and the abolishment of all tendon reflexes. At 19 months of age, the infant was able to move the arms and legs slightly and could hold up its head. The condition improved.

CASE 2.—In an infant, aged 3 months, symptoms were noted at the age of 2 weeks; after that time, voluntary movements, except of the fingers and toes, were absent. The family history was negative. Physical examination showed: a poorly nourished state; enlargement of the abdomen with poor muscle tone; abdominal breathing and general flaccidity of the muscles; hypermobility of the joints; sensation, normal, and abolished reflexes. The infant died at the age of 5 months of bronchopneumonia. Necropsy was not performed.

LOVE, Jacksonville, Fla.

SOME PROGNOSTIC VALUES IN THE MEASUREMENT OF INTELLIGENCE. I. S. WILE, New York State J. Med. **27**:776 (July 15) 1927.

The intelligence quotient is an index of what the child can do at a definite age, what he cannot do, what he had done and what he has failed to do.

Variation of the intelligence quotient under changing conditions is of definite diagnostic and prognostic value. An intelligence quotient in the same child may show marked changes at different times. A girl with marked mental conflicts had a different times quotients of 110, 135, 120, 115 and 130.

In mental deficiency, measurement of the intelligence helps both in the classification and to prognosticate the economic and social levels likely to be reached by the patient.

Lowering quotients in epilepsy, encephalitis, meningitis and in injuries of the head are of serious import.

Numerous cases are briefly reported to show the value of repeated studies of the intelligence in congenital syphilis, spastic paralysis, encephalitis, chorea, hookworm infection, meningitis and other conditions.

Absolute decisions should not be made on one examination, especially in making a diagnosis of mental deficiency. AIKMAN, Rochester, N. Y.

PUBLIC HEALTH REPORTS, U. S. Public Health Service **42**:2487 (Oct. 14) 1927.

State health officers from forty-two states sent in telegraphic reports of 650 cases of poliomyelitis for the week ending Oct. 8, 1927, as against 675 cases reported by forty-four states for the preceding week. For the fourteen weeks from July 3 to Oct. 8, 1927, 5,227 cases were reported as against 1,340 cases for the corresponding period in 1926 and 3,772 in 1925.

TETANUS FOLLOWING SMALLPOX VACCINATION. CHARLES ARMSTRONG, Pub. Health Rep. **42**:3061 (Dec.) 1927.

In the study of postvaccination tetanus over a period of years, the United States Public Health Service found that the source of the disease was contaminated dressings or *Bacillus tetani* either on the skin or accidentally introduced into the lesion later. In all cases of the disease the lesion was covered by a shield or other dressings, in most cases large insertions causing large lesions were made and all cases were from primary takes. The shields cause moist lesions with an ideal medium for the growth of the proteolytic bacteria. Gauze becomes incrusted, and the adhesive straps cause constriction of the tissues when they swell. Large lesions much more easily permit the growth of *B. tetani.*

Experiments on monkeys and rabbits vaccinated with purposely contaminated vaccine bore out the foregoing observations.

The following recommendations are made: (1) a local dressing should not be used, (2) never make an insertion more than one eighth of an inch in diameter, (3) use gentleness in preparing the skin, as abrasions spread the lesion, (4) the insertion of the deltoid is the best site, (5) vaccinate during the first year if possible; older children have little sense of cleanliness and are much more subjected to contaminating dirt.

MUSCLE TRAINING IN INFANTILE PARALYSIS. WILHELMINE G. WRIGHT, Pub. Health Rep. **42**:3113 (Dec.) 1927.

The postfebrile treatment in infantile paralysis consists of prevention of deformities and restoration of the function of the muscle. The former is done by rest and mechanical relaxation of the muscles affected, for instance the prevention of toe drop, sagging shoulder or a stretching of weakened muscles. The problem of restoring the function of the muscle is one usually for the

family physician, in that he must study the muscles affected, and for the parents, in that they must not falter in having the patient keep up the prescribed exercises. The muscle training should start as soon as motion does not cause pain, although premature manipulation is a serious mistake. Contractures should be treated before exercises are started. Muscle training is necessary because muscles or parts of muscles are called on to perform functions which they previously did not have to do as well as to prevent atrophy of the existing tissue by disuse. Massage, heat and passive motion are aids to active motion because they stimulate better circulation; these are best used just before the beginning of the exercises. Increase in exercises should be gradual, and fatigue is especially to be avoided.

A more or less detailed list of exercises for the various muscles is given.

BARNETT, Chicago.

ON THE SIGNIFICANCE OF THE FLEXOR POSTURE OF THE UPPER LIMB IN HEMIPLEGIA, WITH AN ACCOUNT OF A QUADRUPEDAL EXTENSOR REFLEX. W. RUSSELL BRAIN, Brain **50**:113 (June) 1927.

The author comments on the lack of satisfactory explanation of the phenomenon of the flexor posture of the upper limb in hemiplegia while extensor posture of the lower limb has been thoroughly investigated and is fairly well understood. Flexor postures of the fore limb are comparatively rare sequels of experimental lesions of the nervous system. He discusses the flexor posture of the forelimb in anthropoid apes and monkeys and its evolutionary significance and the factors which reflexly influence the posture of the upper limb in hemiplegia: "1. The quadrupedal extensor reflex. 2. Reflex effects of flexion of the lower limbs in the erect and supine attitudes. 3. Tonic neck reflexes. 4. Reflex effects of changes in the position of the body in space; tonic labyrinthine reflexes; and 5. the effect of flexion of the unaffected upper limb." Anatomic evolutionary changes have been "in the direction of making a stable erect posture possible with a minimum of muscular work. There is no necessity for man to use his arms for balancing except when he slips. This increased stability in the erect posture is in process of attainment in the giant anthropoids." Brain then suggests that the plantigrade stage in human evolution was reached with the development of a giant anthropoid able to stand erect with arms which were not needed either for balancing or support in the flexor resting posture. This, he thinks, may explain the association in hemiplegia of the flexed upper with the extended lower limb, which is the primitive human standing attitude. He quotes Sherrington as showing that decerebrate rigidity is reflex quadrupedal standing. He believes that these facts indicate that the hemiplegic posture should be regarded as reflex erect standing.

ON THE METHODS OF EXAMINING THE CONDITIONED FOOD REFLEXES IN CHILDREN AND IN MENTAL DISORDERS. A. G. IVANOV-SMOLENSKY, Brain **50**:138 (June) 1927.

The author states that the work on "Conditioned Food Reflexes in Children" was begun in Russia twenty years ago by Krasnogorsky, who studied the conditioned motor reflexes in the form of opening the mouth and swallowing after associating different optic, acoustic and skin signals with feeding. Miss Mateer has utilized the same method in the United States in examining children. Ivanov-Smolensky describes his apparatus (of which there is a diagram) and his methods including a diagram of records. He says his "experiments have proved that we can distinguish three groups of children: (*a*) children with a healthy nervous activity in whom positive and inhibitory conditioned reflexes are formed quickly and remain stable; (*b*) children with feeble internal inhibition, i. e., those in whom nervous irritation is predominant; (*c*) children in whom cortical inhibition is more prominent than irritation." The author is "investigating the functional value of the cerebral hemispheres, and the peculiarities of age and type of the nervous activity of children aged from 5 to 15 years."

and he has also applied his methods "in examining the conditioned reflexes of some categories of individuals in imbecility, mental debility, schizophrenia and general paralysis." By his methods, the grasping conditioned reflex of great biologic importance can be examined.

FORCED GRASPING AND GROPING. W. J. ADIE and MACDONALD CRITCHLEY, Brain **50**:142 (June) 1927.

There are so few unequivocal signs of disease confined to the frontal lobes of the brain that the diagnosis of frontal tumor is rarely clear cut. Adie and Critchley report three cases of frontal tumor of one lobe in adults. A common feature in their cases is the "presence of involuntary grasping and groping movements in the limbs on the side opposite to the frontal lobe in which a tumour was found." Case histories and movements in detail are given. The literature is reviewed and a chart of similar cases is shown. The authors discuss the nomenclature, the site of the lesion producing forced grasping and groping, the relation to delay or difficulty in relaxing the grasp in other conditions, and the nature of forced grasping and groping. They state that "a consideration of the stages in the development of grasping in normal infants gives a clue to the manner in which this function might be expected to disintegrate in disease, and provides us with examples of imperfect grasping analogous with those that concern us here." The different stages in development up to a year of age are described. The authors show that defects strictly analogous with those in their patients are seen in infants learning to use their hands and that "the movements during this learning process become less automatic and more controlled as reflexes are acquired conditionally which supplant the unconditioned reflexes dependent on reflex paths present at birth." They conclude "that the anatomical basis for acquired (conditioned) reflexes is in the cerebral cortex; when the part of the cortex containing the anatomical paths concerned in the conditioned reflexes associated with the function of grasping is damaged, more primitive, less controlled reactions in the shape of forced grasping and groping reappear."

SUBCORTICAL EPILEPSY. WILLIAM G. SPILLER, Brain **50**:171 (June) 1927.

This is Dr. Spiller's presidential address read before the Philadelphia Neurological Society, Jan. 28, 1927. He discusses one particular form of convulsive attack as exemplified by two adult patients under his observation. The "prominent symptoms were brief tonic spasms of both hands and one lower limb in one case, and of one upper and one lower limb on the same side in the other." In neither case were clonic seizures present. Search for conditions in the literature allied to the tonic attacks, without the clonic movements presented here, involved a review of experiments in decerebration and subcortical pathologic conditions. The functions of the striate body are imperfectly understood, yet the nomenclature has increased beyond the proved basis. Although the postencephalitic syndrome would point to a special segmental representation in the corpus striatum, experiments with the faradic current have not demonstrated this. The reports of Foerster are cited at length, and contrary views are mentioned. In conclusion, it is intimated that the phenomenon under discussion may be interpreted on the basis of subcortical pathologic changes, possibly in the corpus striatum.

MICROCEPHALY AND DIFFUSE GLIOSIS: A CLINICO-PATHOLOGICAL STUDY OF FOUR CASES. WALTER FREEMAN, Brain **50**:188 (June) 1927.

Diffuse sclerosis of the brain still presents a number of unsolved problems, and Freeman believes that while various types have been separated from the main condition, yet there is considerable overlapping in those remaining. He states that while "cerebro-macular degeneration (Tay-Sachs disease), tuberous sclerosis and Schilder's encephalitis periaxialis have been well defined by

various authors, there is still confusion in regard to the anatomic delineation of the Westphal-Strumpell pseudosclerosis, diffuse gliosis as such, and the association of a partial diffuse gliosis with microcephaly." According to the author, these three conditions may be slightly different manifestations of the same conditions, either taking place at different stages of development or affecting different parts of the central nervous system. He reports four cases, in which the patients were aged 3 and 8 months and 3 and 6 years, which were of considerable clinical and great pathologic similarity, and the outstanding features of which were microcephaly, generalized muscular rigidity with tonic fits and a peculiar, almost noiseless, cry and severe idiocy. Freeman summarizes his article as follows:

"Pathologically, there is found enormous diffuse fibrillar gliosis of parts of the spinal cord, medulla oblongata, pons and mesencephalon, sometimes involving also the basal ganglia. Aside from this there is little structural alteration at these levels. The cortex and basal ganglia give evidence of defective development from a cytological and architectural point of view. Disordered fat metabolism is a distinctive feature. Even though the cells and myelin sheaths appear relatively normal, there is a large amount of fat scattered dust-like through the gray matter, both within cells and outside and the collection of small masses of this fat in ordinary compound granule cells about the walls of vessels. The absence of inflammatory, vascular, and well-known degenerative changes is a striking feature."

LUCAS, San Francisco.

A CASE OF ANTERIOR POLIOMYELITIS. WALTER BROADBENT, Proc. Roy. Soc. Med. **20**:72 (Oct.) 1927.

Broadbent reported a case in a girl, aged 3½ years. The patient had been ill for a fortnight before admission to the hospital with congestion of the left upper lobe of the lung. Ten days later she vomited and complained of headache, and a squint was noticed. The next day she was drowsy and presented a picture very much like that of a commencing meningitis. The spinal fluid was clear and not under pressure. It contained a trace of albumin and 15 cells per cubic millimeter. The lymphocytes numbered 92 per cent and the polymorphonuclears, 8 per cent. Methenamine was administered. The child improved in a general way in the next week or two, but paralyses of the left shoulder girdle and muscles of the neck developed. It was then evident that the condition was poliomyelitis and not meningitis.

WILLIAMSON, New Orleans.

THE AFTER-CARE OF INFANTILE PARALYSIS. KING, Rev. internat. de l'enf. **4**:661, 1927.

Without complete and continuous cooperation of parents and physician in the after care of this disease, the best results cannot be obtained. Miss King reviews the accepted opinion regarding the management of cases in the acute and convalescent stages. There are three therapeutic measures employed directly in securing muscular improvement after all pain and tenderness have disappeared. The first of these is massage, which must be intelligently directed and properly controlled. Electric treatments are not approved. By far the most important agent is muscle training, which seeks to establish new routes for the nerve impulses. In order to employ this advantageously, a complete preliminary survey is necessary, with the patient stripped and the integrity of all the voluntary muscles noted. From this survey a suitable program is mapped out, to be carried on daily, each exercise to be done not more than ten times. If the child has little power in its muscles, immersion is recommended in a warm saline bath in which the bouyancy of the water enables the patient to execute movements not possible on the table. If contractures have developed, these must be corrected before muscle training can be used to advantage. Many of the earlier cases can be relieved by manipulation:

the older cases may be treated with suitable casts. Braces and other apparatus are not desirable on a weakened leg, but in conjunction with massage and muscle education, they have an important place. They are to be considered as protective and conservative rather than therapeutic. The same can be said of corsets in the management of spinal curvature.

AMESSE, Denver.

SPIROCHETAL PURULENT MENINGITIS. E. FANTON, Clin. Pediat. **9**:582 (July) 1927.

A case of spirochetal meningitis is reported in a boy, aged 12 years, who suffered for many years from a chronic otitis media. The onset and subsequent course to a fatal termination were typically that of the ordinarily observed meningitides. Two spinal punctures were performed, and a purulent fluid was obtained each time. On bacteriologic examination the second specimen showed the presence of a spirochete by special staining. The exact type to which the spirochete belonged was not determined, due to lack of material. The author believes that it reached the meninges through the chronically infected ear.

ZAMBARANO, Northville, Mich.

FATAL ACUTE ASCENDING PARALYSIS FOLLOWING THE USE OF ANTIRABIES VACCINE IN A CHILD BITTEN BY A RABID DOG. PELFORT, CONRADO, Arch. Americanos de med. **3**:109, 1927.

The case concerns a child, aged 2 years. Ten days after the injection of the vaccine, pains developed in the pelvis and lower extremities, with loss of function in the latter and eventually complete paralysis. The sphincters were not involved. Death ensued five days after the onset of the symptoms, following complete loss of consciousness and marked hyperpyrexia. The picture resembled closely that seen in Landry's paralysis.

SCHLUTZ, Minneapolis

NEUROSIS OF THE VEGETATIVE SYSTEM IN THE SMALL CHILD (FEER). HANS MÜLLER, Arch. f. Kinderh. **81**:81 (June) 1927.

Under the title, suggested by Feer, the author gives a brief discussion of acrodynia, and describes seven new cases observed by him in Zurich.

TUBERCULOSIS VERRUCOSA CUTIS CAUSED BY THUMB SUCKING. J. DUKEN, Arch. f. Kinderh. **81**:100 (June) 1927.

Tuberculosis of the skin is common in childhood, but tuberculosis verrucosa cutis is usually regarded as occurring almost exclusively in adults. An infant, aged 4 months, with pulmonary tuberculosis was admitted to the hospital and four weeks later developed lesions on the thumb typical of tuberculosis verrucosa cutis. Removal of one of the lesions by biopsy confirmed this diagnosis. The child subsequently developed tuberculous cervical glands and papulonecrotic tuberculids.

HEMPELMANN, St. Louis.

EYE REFLEX IN NECK IN EARLY INFANCY. ALBRECHT PEIPER, Jahrb. f. Kinderh. **113**:87 (June) 1926.

To demonstrate the reflex, Peiper brings the child to a sitting position so that the head, which is not supported, is approximately in equilibrium, and suddenly throws light into the eyes. The child reacts by drawing his head back. The musculature of the back takes part in the movement, so that the whole body goes into opisthotonos. If the experiment is repeated while the head is in this position, there is a further slight movement backward. The reflex has obviously the same purpose as shutting of the lids and narrowing

of the pupils; that is, to protect the retina from too strong a light. When the light is attached to the forehead, the head of the child remains bent back as long as the strong light is there. The reflex, therefore, has tonic qualities. It is a function of the brilliance of the light and may be elicited for several months. It disappears if the general condition is poor. The reflex is present in young premature infants weighing about 1,500 Gm. In normal, full-time children the reflex disappears earlier than in premature infants.

CZERNY'S PHENOMENON EXAMINED ROENTGENOLOGICALLY IN CHOREA MINOR. OSKAR GÖTTCHE, Jahrb. f. Kinderh. **113**:90 (June) 1926.

In fourteen cases of manifest chorea, Göttche was able to demonstrate the typical behavior of the diaphragm by means of the fluoroscope. In two cases he diagnosed chorea in the beginning of its course before the appearance of motor disturbances. Later, the Czerny phenomenon, which had been seen with the fluoroscope, was demonstrable clinically.

PACE, Chicago.

SYRINGOMYELIA IN CHILDHOOD. E. J. BERMAN, Monatschr. f. Kinderh. **37**:1, 1927.

Berman reports a case in a girl, aged 11 years, and reviews the previous literature. The disease is rare during the first ten years. Etiologically, most authors consider it a hereditary anomaly in the development of the spinal cord, but this theory has not been proved. The clinical picture is the same as that in adults, except that the condition is a little more difficult to diagnose in children. Prognosis is unfavorable, roentgen-ray or radium therapy having been unsuccessful.

SPASMUS NUTANS AND NYSTAGMUS. G. KNOLLER, Monatschr. f. Kinderh. **37**:21, 1927.

In view of the fact that these conditions affect children who have spent much time in darkened rooms, the author tried the effects of artificial sunlight. To his surprise he obtained excellent results in all but three of nine cases. Of the three patients who did not respond, two were aged 5 and 7 years and probably showed marked pathologic changes. The third did not complete the treatment.

THE PSYCHOLOGY OF CHILDHOOD. JOHANN SUSSMANN GALANT, Monatschr. f. Kinderh. **37**:97, 1927.

Galant gives a review of the writings of a number of authors such as Tolstoy, Karl Spitteler, Freud, Binet and others with extracts from reminiscences of their early childhood. These brief quotations are concerned with the development of the ego, morality, dream life, sadism and play. Play is considered a biologic necessity, and to the child represents reality. As soon as the child realizes the difference between the fantasy of play and reality, the play, as such, no longer exists.

MENINGEAL PERMEABILITY IN CHILDHOOD. W. A. LEONOW, Monatschr. f. Kinderh. **37**:112, 1927.

Several years ago, Jervell recommended the administration of uranium in doses of 0.03 Gm. per kilogram of body weight as a test for meningitis. Within three hours, the uranium should appear in the spinal fluid as a definite green fluorescence in cases of meningitis. Using this method the author tested children suffering from a number of different conditions. He found that meningeal permeability in normal children is increased over that in adults, but he does not believe that this entirely accounts for the development of meningismus or meningitis. It does play a rôle, however. In exudative

diathesis and chronic nutritional disturbances, there is no change. In rickets and spasmophilia, a slight increase in permeability takes place. In pneumonia, acute and chronic infections and invasion with ascaris, there is sufficient increase to permit the passage of toxins. Uremia has a marked influence. The permeability is particularly injured in meningitis. In the latter case, foreign substances pass from the blood to the spinal fluid much more rapidly than they are excreted from the fluid to the blood.

FAMILIAL PROGRESSIVE CEREBRAL DIPLEGIA WITH CONGENITAL CATARACT. OSCAR HERTZ, Monatschr. f. Kinderh. **37**:135, 1927.

Hertz reports two cases occurring in only children of two sisters.

GERSTLEY, Chicago.

## Diseases of the Ear, Nose and Throat

STUDIES IN STUTTERING. SAMUEL T ORTON, Arch. Neurol. & Psychiat. **18**:671 (Nov.) 1927.

In an introduction to the following article, the author describes a case of stuttering which developed after coercion of a left handed child to write with his right hand. These and other cases would seem to support the theorem that stuttering is often an expression of confusion in cerebral dominance.

STUDIES IN STUTTERING. LEE E. TRAVIS, Arch. Neurol. & Psychiat. **18**:673 (Nov.) 1927.

Comparisons were made between six persons with severer forms of stuttering and several normal speakers. The records of normal speech show an integration of the various units of the breathing mechanism.

The records of stuttering show a disintegration of certain of the motor speech units which is apparent at various times by: (1) a complete antagonism between the action of the thorax and that of the abdomen; (2) a marked synchronism between the movements of the larynx and those of the various units of the breathing apparatus; (3) a marked prolongation of inspiration; (4) large vertical movements of the larynx during inspiration; (5) clonic and tonic spasms of the various muscles of speech production, and (6) the apparent introduction of a new rate of tremor in the abdomen.

INFANTILE MASTOIDITIS WITH GASTRO-INTESTINAL SYMPTOMS. H. W. LYMAN, Arch. Otolaryng. **6**:526 (Dec.) 1927.

Operations on the mastoid were performed on forty-three infants under 2 years of age in the St. Louis Children's Hospital. Of these cases, thirty presented marked gastro-intestinal symptoms, with eight deaths, and thirteen did not present any gastro-intestinal symptoms, with one death. This fatality was due to causes entirely extraneous to the infections in the ear.

These figures show forcibly that mastoid infections in infants with gastro-intestinal symptoms are much more serious than in those without, and that prompt and thorough operation is much more important in these cases than in the usual mastoid abscess.

Seventeen different organisms were found in the various cultures, with *Streptococcus hemolyticus* predominating. These different organisms did not show any special relationship to the gastro-intestinal symptoms, all of the more common varieties occurring in cases both with and without gastro-intestinal disturbances. So, while it may be possible that *Streptococcus hemolyticus*, or some special strain of that organism, is a special etiologic factor in the production of gastro-intestinal symptoms in these cases, it is certain that many other organisms can produce the same effect.

THE PATHOLOGY OF MASTOIDITIS IN INFANTS. BERNARD J. MCMAHON, Arch. Otolaryng. **7**:13 (Jan.) 1928.

In a series of thirty-nine cases of mastoiditis in infants occurring from September to May, twenty-two, or 56.4 per cent, showed the symptom complex of increased temperature, diarrhea, vomiting, loss of weight and athrepsia. This symptom complex has the following effects on the prognosis: 1. The prognosis is bad if, when edema of the mucosa of the mastoid antrums is found microscopically, the diarrhea-vomiting complex is present. 2. The prognosis is good when fibrosis of the mucosa of the mastoid antrums is found microscopically, even though the symptom complex is present. 3. When the diarrhea-vomiting symptom complex is not present, the prognosis is equally good whether the mucosa of the mastoid antrums shows edema or fibrosis microscopically.

SANFORD, Chicago.

NASAL DISEASE IN RELATION TO ASTHMA. SIR JAMES DUNDAS-GRANT, Practitioner **119**:341 (Dec.) 1927.

Working clinically from Dixon's experiments in which irritation of the nasal mucus membrane in decerebrate animals brought about contraction of the bronchial muscle, the author had gratifying results in a series of patients with asthma by removing enlarged middle turbinate bones which pressed on the septum. Even in his allergic patients, improvement was marked, although the allergy persisted.

GELSTON, San Francisco.

THE PRESENCE OF STREPTOCOCCUS HEMOLYTICUS (SCHOTTMÜLLER) IN THE PHARYNX AND ON THE TONSILS IN APPARENTLY HEALTHY INDIVIDUALS. München. med. Wchnschr. **73**:2117 (Dec. 10) 1926.

This study was based on five groups of patients: (1) 206 children in a boarding school in which scarlet fever had been present; (2) 80 children from an orphanage; (3) 110 apparently healthy pupils from another school; (4) 17 healthy adults, and (5) 100 patients suffering from various disorders of the throat and nose. Those of groups 1 and 2 who showed *Streptococcus hemolyticus* were reexamined later. Group 4 was examined repeatedly. In group 1, 7.7 per cent showed *Streptococcus hemolyticus;* in group 2, 16.2 per cent; group 3, 7.3 per cent; group 4, 5.8 per cent, and in group 5, 26 per cent.

CEPHALOCELE INTRANASALIS. WILHELM EDEL, Ztschr. f. Hals-, Nasen- u. Ohrenh. **19**:163 (Aug. 27) 1927.

A boy, aged 18, was said to have suffered from nasal polyps soon after birth. These were removed first when he was 9 months old. A continuous watery discharge from the nose was reported. Polyps were removed repeatedly, until a greater intervention under general anesthesia, during his fourth year, gave the patient relief. He suffered frequently from severe headache, especially above the nose. During these attacks, he acted dishonestly and even committed burglaries, whereas at other times he was industrious, kind hearted and intelligent. Examination revealed the fact that the left side of the nose was almost completely filled with a grayish-white smooth tumor with a shining surface. The roentgenogram showed absence of frontal sinuses and cloudiness of both ethmoids and of the left maxillary antrum. A portion of the tumor was removed for examination. A probe, carefully introduced, entered the cranial cavity without resistance. The specimen contained tissue of the central nervous system with a covering of connective tissue. Five days later, the patient complained of headache; six days later, in addition, he suffered pain while swallowing, and, during the following night, he vomited. The next day, the patient was restless and dazed, and showed a positive Kernig sign. An

operation was performed according to the Jansen-Ritter technic, and the frontal lobe of the brain was elevated in order to reach the sac of the hernia, which was extending into the nasal cavity through an opening, 1.5 by 1 cm., in the lamina cribrosa. The sac was joined to the bone at its posterior border and also to the septum. Respiration became difficult, and the patient died on the operating table. Necropsy was not permitted.

Intranasal encephaloceles have been observed by Richter, Cruveilhier, von Meyer, Fenger, Kayser, Schoetz, Nager, Froboese and Lenhoff. Only one patient (Fenger's) or, perhaps, two (Lenhoff's), survived an operation. Nevertheless, surgical intervention is contraindicated. The diagnosis is difficult. Only Nager diagnosed his case without operation; he, however, made a trial puncture, a procedure which might not always be harmless. One should consider the possibility of intranasal encephalocele and resort to trial puncture rather than to trial excision or trial snaring if the anamnesis, appearance, compressibility, transillumination, roentgen-ray examination and irrigation of the maxillary sinus do not lead to a diagnosis.

AMBERG, Detroit.

## Diseases of the Skin

TWO CASES OF FOLLICULITIS ULERYTHEMATOSA RETICULATA (MACKEE AND PAROUNAGIAN) COMBINED WITH LICHEN SPINULOSUS, EPIDERMAL CYSTS, AND FOLLICULITIS DECALVANS. H. W. BARBAR, Proc. Roy. Soc. Med. **20**:176 (Dec.) 1927.

Two patients — a brother and sister — were of exceptional interest because in both were combined conditions which have been separately described and which have excited considerable discussion as to their significance and relationships. On the cheeks and temples in both patients was seen the peculiar follicular pitting described by MacKee and Parounagian (*J. Cutan. Dis.* **36**:339, 1918) as "folliculitis ulerythematosa reticulata." In both, but particularly in the sister, were large numbers of epidermic cysts exactly similar to those seen in histologic sections from MacKee and Parounagian's patients. In these cases some of the cysts were as large as a large pea, whereas in MacKee and Parounagian's patients they were apparently microscopic. On the limbs and trunk were follicular horny plugs, which varied in appearance and degree from those seen in ordinary keratosis pilaris to the filiform spines of so-called lichen spinulosus. In certain situations on the limbs and trunk, there was follicular atrophy which resulted in deep pits. On the scalps of both patients, there was cicatricial atrophy with the production of bald areas on the vertex. It will thus be seen that in this brother and sister were follicular lesions involving practically all parts of the body in which pilosebaceous follicles exist (the palms and adjacent part of the anterior surface of the wrist were entirely free); these lesions comprised horny plugging, lichen spinulosus, formation of epidermic cyst, atrophy with pitting, and bald areas on the scalp. The cases thus bring into line the follicular atrophy of the face described by MacKee and Parounagian, and the syndrome described by Graham Little, Dore, Wallace Beatty and Speares and others of lichen spinulosus and folliculitis decalvans.

GUY, Pittsburgh.

A STUDY OF RINGWORM INFECTION IN HORSES AND IMMUNITY IN EXPERIMENTAL RINGWORM. BROCQ-ROUSSEU, A. URBAIN and J. BAROTTE, Ann. de l'Inst. Pasteur **41**:513, 1927.

The only organ susceptible to infection by the ringworm parasite (usually a microspore) is the skin. Immunity is readily established with living cultures and is in proportion to the extent of the lesion of the skin. Antibodies are easily demonstrable by complement fixation in infected guinea-pigs and to a lesser degree in horses though the reaction is specific. Trichophytin (similar to tuberculin) gives positive skin reactions in 100 per cent of infected animals.

Tuberculin also gives positive skin reactions in some of these cases. Some tuberculous persons and animals show positive intradermal reactions with trichophytin.

G. B. RHODES.

LICHEN PLANUS IN INFANCY. M. G. PETGES, Bull. Soc. franç. de dermat. et syph. **34**:515, 1927.

In about 5,600 cases of diseases of the skin in children, Petges has encountered 10 cases of lichen planus. The youngest child in this series was 6½ months old. Study of these cases added nothing to the present knowledge of the pathogenicity of the disorder. In children, lichen planus seems to occur oftener than in adults in the acute form and to run a shorter course.

A CASE OF LICHEN PLANUS STRIATUS OF RADCLIFFE CROCKER. J. M. PEYRI, Bull. Soc. franç. de dermat. et syph. **34**:517, 1927.

In a boy, aged 3, a papular eruption developed on the back of the right hand, with subsequent development of similar lesions in a linear arrangement on the arm. The case corresponded to that described by Crocker as lichen planus striatus.

A CASE OF "CUTIS LAXA." M. RASPI, Riv. di clin. pediat. **25**:648, 1927.

A case is reported of loose skin ("cutis laxa") in a child, aged 13 months. The child was born with a marked edema; when the edema subsided, the looseness and folding of the skin were first seen.

HIGGINS, Boston.

REVIEW OF THE LITERATURE ON SKIN DISEASES IN CHILDREN DURING 1926. CARL LEINER, Monatschr. f. Kinderh. **37**:224, 1927.

Leiner presents a complete review with the thoroughness and clearness of one well versed in the subject.

GERSTLEY, Chicago.

THE REACTION OF THE SKIN IN CHILDHOOD TO DIFFERENT IRRITANTS. L. ADELSBERGER, Ztschr. f. Kinderh. **43**:373 (May) 1927.

Eczema is perhaps no longer to be looked on as a result of metabolic disturbances which work themselves out on the skin but as the result of special capacity for reaction of the whole organism, particularly of the skin. Bloch believes that two factors are necessary for the appearance of eczema: an irritant and a skin sensitive thereto. Adelsberger made tests on the skin of children of different ages, susceptible and nonsusceptible to eczema, to find how they reacted to irritants placed on the skin and to foreign proteins by intracutaneous injection. Physiologic sodium chloride solution, horse serum and caseosan were used for injections and only mild irritants, such as water, tincture of arnica and quinine, to which normal skin are indifferent, were used superficially. The small child and the older child do not differ from adults in regard to skin irritants. The new-born infant and the infant in the first months of life react to irritants causing eczema, and to intracutaneous irritants with difficulty and only occasionally. Their skin, however, is more sensitive than it is in the later months to mechanical injuries to which they react with an intertrigo. It seems that these alterations in the skin cannot be identified with the eczema of adults and that a special anatomic quality of the skin is responsible for their appearance, but not in the sense of an idiosyncrasy. The eczema of the young infant is not a real eczema but is a change in the skin conditioned by the processes of desquamation and maceration. The infant in its second half-year, however, reacts to intracutaneous irritants and especially to irritants causing eczema in a marked manner. In most cases they react to different irritants simultaneously. Since the increased susceptibility to skin irritants occurs with an increased susceptibility to other irritants, such as

bacteria, it is apparent that changes in allergic condition affect the organism of the older infant more easily and more frequently. The organism is altered, especially the skin, which is made ready for the appearance of eczema.

WHITE DERMOGRAPHISM IN CHILDREN. A. S. SOKOLOW, Ztschr. f. Kinderh. **43**:391 (May) 1927.

Red dermographism, according to Sokolow, appears in childhood with some regularity, varying with the condition of the skin, the constitution, the age and the presence of other diseases. In the first year it disappears in a small number of cases, and white dermographism is established. Assuming that white dermographism in exudative diathesis reveals a tendency of the skin elements to constriction, the author thinks that he may be able to explain some constitutional anomalies such as prurigo and urticaria. Many years ago, Unna and Joseph noticed a disturbance of equilibrium in the contraction of the smallest vessels of the skin which might lead to a diminution of outflow, and Mautner also saw in urticaria a contraction of the smallest veins. Sokolow suspects that the usual pallor of the skin in exudative diathesis and perhaps also the eczematous processes are concerned with this tendency to constriction. Further study from this point of view may give some light on the origin of some disturbances in children with exudative diathesis.

CLINICAL PICTURE AND ETIOLOGY OF WIDESPREAD INFECTION WITH TRICHOPHYTA AND MICROSPORA: EXANTHEM. LICHEN TRICHOPHYTICUS (JADASSOHN-BLOCH) AND LICHEN MICROSPORICUS (ARZT-FUHS). R. BRAUDE and M. PER, Ztschr. f. Kinderh. **43**:525 (May) 1927.

The sharing of the whole apparatus of immunity in fungus disease of the skin was confirmed by Jadassohn in 1911. He was the first one who described children with a deep infection with trichophyticus of the hairy portion of the scalp, accompanied by a disseminated licheniform exanthem on the body which he put in direct connection with the process on the head.

Four cases are here reported from which the authors draw these conclusions: not only kerion celsi, kerion microsporicum and trichophytia media, that is, an intermediate form of trichophyta but superficial forms of trichophyta and microspora of the hair-covered scalp may cause secondary generalized exanthems which occur as phenomena of partial immunity. Like physical and chemical irritants, roentgen rays sometimes constitute the points of origin of infection with trichophyta and microspora which must be viewed as the expression of an allergic reaction of the skin shown partly by its increased sensitivity to the intradermal injection of fungus extract. Lichen trichophyticus and microsporacus are sometimes found in rashes occurring simultaneously; Lichenoid-eczematoid and papulo-vesiculo-pustular. The culture of scales of lichenoid efflorescence yielded a growth of a fungus of the type of *Microsporon lanosum*. In the cases observed by the authors there could be observed a reaction to specific fungus extract and at the same time a positive reaction to the extract of a related fungus; that is, positive epidermo mycotic group reaction. The blood picture in secondary exanthems was characterized in three cases by a leukocytosis and eosinophilia, and in one case there was found a stimulation cell of Türk.

THE INCUBATION TIME OF ERYTHEMA NODOSUM. ARVID WALLGREN, Ztschr. f. Kinderh. **43**:543 (May) 1927.

In 1922, Wallgren reported the simultaneous occurrence of erythema nodosum in children of two families with tuberculosis and referred to observations made elsewhere on the epidemic occurrence of the disease. By a consideration of the four examples here reported, the author believes that he has some support for the assumption that the incubation period of erythema nodosum is identical with the incubation period of tuberculosis, and that erythema nodosum is in reality a skin manifestation of tuberculosis.

EMERSON, Boston.

## Surgery and Orthopedics

FREIBERG'S INFRACTION OF THE METATARSAL HEAD. (KOEHLER'S METATARSOPHALANGEAL SYNDROME: JUVENILE DEFORMING METATARSOPHALANGEAL OSTEOCHONDRITIS). KEITH T. MEYER, Am. J. Roentgenol. **16**:20 (July) 1926.

This condition has been reported only about seventy-five times in the literature. The etiology is unknown and the symptoms include pain, definitely localized over the site of the affected bone, and sensitivity to pressure over the head of the involved metatarsal bone. Diagnosis is made by means of the roentgenogram, which shows (1) broadening of the metatarsophalangeal joint space; (2) flattening of the neck and distal portion of the shaft, with the resultant disappearance of the constriction at the site of the neck, and (3) changes in bone absorption in the head suggestive of sequestration. In addition, in this reported case there was a loss of circular form and irregularity of the articular surface of the proximal phalanx. Diathermy applied daily over the site of tenderness for a period of two weeks and restraint from walking on the foot resulted in a symptomatic cure.

STULIK, Chicago.

NEUROSURGERY IN THE NEW-BORN AND CHILDREN. WILLIAM SHARPE, New York State J. Med. **27**:701 (July 1) 1927.

The author finds acute injuries of the brain to outnumber any other conditions of the nervous system calling for surgical treatment. While the automobile is the most common cause, falling on the head from a height of trivial extent, 2 or 3 feet, frequently causes severe cranial injury.

If shock is present all extensive neurologic examinations, roentgen-ray examination, etc., should be postponed, and all attention directed toward overcoming the shock.

The expectant palliative treatment by warmth, quiet, ice helmet, saline solution for dehydration and spinal drainage by repeated spinal punctures has been successful in 71 per cent of these patients, and it is only in 16 per cent of the entire series of 217 cases that cranial drainage by decompression was indicated and performed. The advisability of operative treatment in adults is twice as common (29 per cent.).

Operative treatment for cranial drainage is used when expectant palliative treatment fails to lower the intracranial pressure as estimated by the spinal mercurial manometer. The mortality for operative treatment is not given.

In 500 consecutive new-born children, irrespective of the type of labor, bloody spinal fluid was found in 9 per cent. Only three of these children died.

Absorption of the blood occurs in the mild cases, but with larger hemorrhages early repeated lumbar punctures are of greatest value.

*Hydrocephalus.*—Three fourths of the author's cases were of the external type. Successful drainage of external hydrocephalus is encouraging, but is discouraging for internal hydrocephalus.

*Spina Bifida and Meningocele.*—Increased intracranial pressure was found in 86 per cent. This produces a protrusion of the meninges at the site of weakest closure in the cranial or cerebral canal. In 47 per cent of the infants, hydrocephalus was found and hydrocephalus developed in 58 per cent of postoperative cases. Operation is advised within the first three months after birth.

*Brachial Paralysis at Birth.*—If marked improvement is not shown in from three to six months, operative repair should be attempted. Even with early operation complete recovery does not occur.

AIKMAN, Rochester, N. Y.

PHRENICECTOMY IN CHILDREN. A. MAGRASSI, Clin. Pediat. **9**:611 (Aug.) 1927.

The author performed this operation in four children, aged 22 months, 5, 6 and 7 years, respectively. In one child with tuberculous empyema on whom resection of the rib was performed on two occasions, the phrenic nerve

was avulsed, but after twenty-four days the patient developed a tuberculous meningitis and died. In another patient, the same operation was performed for a persistent pleural fistula of one year's duration. The patient was discharged as cured after fourteen months. Phrenicectomy was done in another patient with a persistent sinus following a left-sided nontuberculous empyema. A clinical cure was obtained after eleven months. In the fourth patient, a phrenicectomy was done, with favorable results after seven months. This patient had a left-sided purulent pleurisy, with cavitation in the upper lobe. Following the operation there was complete immobilization of the left hemidiaphragm, and signs of cavity were not present in the upper lobe.

The author believes that phrenicectomy is often indicated in children. Sometimes it may be difficult to find the nerve if there is much cervical adenitis in the lateral regions of the neck. Tuberculous or nontuberculous pleural fistulas resistant to other forms of surgical therapy, may be considerably benefited by this operation. Cavities in the upper lobes are frequently obliterated following phrenicectomy. This was demonstrated in the fourth case reported. In some cases, the function of the diaphragm is resumed at variable periods after the operative procedure. This is usually due to the presence of an accessory phrenic nerve. Occasionally, a simple phrenicotomy will suffice in cases of fistula of long standing.

ZAMBARANO, Northville, Mich.

APPENDECTOMIES IN CHILDREN. A. NEHRKORN, Arch. f. klin. Chir. **144**:559, 1927.

Owing to the fact that the literature on appendicitis deals sparingly with appendicitis in children, the author feels that without doubt many children under 3 or 5 years of age constitute a special class, though appendicitis at this age is not a frequent occurrence. Some authorities are of the opinion that infants are spared from appendicitis, and that when such a diagnosis is made it is a mistaken one, even though pathologists have found a generalized peritonitis which has as its source a perforated appendix.

Diagnosis of appendicitis is difficult, because children give little aid from a subjective standpoint. This applies to children up to 10 years of age. Rigidity is not constant, though vomiting is invariably present, but its significance is slight because of the frequency and ease with which vomiting occurs in children. Diarrhea occurring in association with enteritis, which may easily involve the appendix, is significant. Fever is always present as are leukocytosis and frequent urination, and on rectal examination, either tenderness or a palpable mass, or both, are found on the right side. Finally, the author states that, in spite of these symptoms, there is not a definite criterion for the diagnosis of appendicitis.

The treatment is always surgical, and in case of doubt, the author advises operation because of the slight risk attending an exploratory laparotomy in modern surgery. In view of the fact that appendicitis reaches its maximum development in considerably less time in children than in adults, earlier surgical intervention is even more imperative than in adults. Intoxication from the infection likewise comes on earlier and apparently is more severe. The greater tendency in children for early perforation may be explained in part by the fact that they have a shorter and more delicate great omentum, and it is in the "walling off" factor that the safety of the child lies. The experience of surgeons has been that operation during the first two days after the onset of the attack affords good results, but that if the attack has persisted for more than two days, conservative treatment is advised to permit a walling off, and then drainage is instituted. The author had a series of thirty patients who belonged in the latter group, and all recovered. Mortality in children under 5 years of age is considerably higher than in those between 5 and 15 years, after which the mortality slowly increases. In the light of his experience, the author is emphatic in his views that early operation is the method of

choice, but that if this has not been possible, one should await walling off of the abscess and then open and drain it.

THE OPERATIVE TREATMENT OF CONGENITAL CLEFT PALATE. G. TSCHMARKE, Arch. f. klin. Chir. **144**:697, 1927.

The author reviews forty-five cases of cleft palate in which operation was performed by Langenbeck's method in the Leipzig Clinic during the ten year interval from 1914 to 1924. He divides his material into three groups.

In the first group of twenty-three patients, from 2 to 6 years old, fifteen had involvement of the soft palate, four of both the soft and the hard palate, and four, a complete cleft palate and harelip. The operative results were: one death from anesthesia, six failures, two partial failures and the remaining fourteen successful. In a "follow-up" of the sixteen patients with partial and complete healing, only nine were located: five of these had distinct improvements in speech, three had practically a complete return of speech, and one, a complete return. These results were obtained without any special teaching in speech.

In a second group of sixteen, between the ages of 7 and 14, five had involvement of the soft palate, five of both the soft and the hard palates, and five of the complete palate, four of which had an associated harelip. The results were anatomically successful in eleven; partially successful in one and a complete failure in four. In a "follow-up" of ten, one was functionally normal, two had made a satisfactory recovery and seven showed some improvement.

In a third group of six, whose ages were 14 and over, one had involvement of the soft palate, four of both the hard and the soft palates, and one had a complete cleft palate and harelip. The operative results in four were successful, in one partial and in one a complete failure. In a "follow-up" of three, two were practically normal functionally, and one was completely normal.

According to the author, the anatomic results compare favorably with those of other authors, showing complete recovery in 64.3 per cent, partial recovery in 12 per cent and complete failure in 24 per cent. As far as functional return in speech is concerned, 9.1 per cent had a complete return, 57.6 per cent showed some improvement and 33 per cent did not show any change. Some authors report better results than these, which Tschmarke attributes to two factors: (1) The anatomically healed operation is not necessarily a physiologic one and (2) a physiologicanatomic healing, because of certain factors, does not always insure a complete functional return. Special teaching of articulation and speech may have an important influence. A great essential is an adequate soft palate for function.

In a summary of the author's observations and those of other investigators, there is only 70 per cent of complete anatomic healing, 25 per cent of partial healing and in 5 per cent, none at all. This is based on Langenbeck's method. Comparatively speaking, the functional return is much poorer than the anatomic healing. The author attributes this to failure in the reconstruction of a functional soft palate and improper postoperative management. As a result he pleads for a better technic that prevents tearing of the sutures, cough, crying and impairment of the blood supply of the muscle flaps.

OPERATIVE TREATMENT OF PYLOROSPASM IN INFANTS. THE LATER GROWTH AND DEVELOPMENT OF SURGICALLY CURED CHILDREN. ARNOLD JACOBSON, Arch. f. klin. Chir. **145**:189, 1927.

Congenital pyloric stenosis belongs to that group of conditions in which the question of conservative management or operation arises. Those opposed to surgical treatment do not regard the operation as a procedure of life or death, and further, they admit that many pylorospasms respond to medical management. As far as operative mortality is concerned, some methods have shown a high mortality, but the method of Ramstedt lends itself to the best results.

The author reports a series of fifty-six cases from Hildebrand's clinic, with a mortality of 38 per cent. He attributes this in part to the moribund condition of the children when brought to the clinic. Since 1923, the mortality has been reduced to 12 per cent, which he attributes to the operative method of Ramstedt. The mortality is also dependent, to some extent, on the condition at operation, and as to whether or not the pyloric condition is a spasm instead of a true hypertrophy. The longer the delay in operation when the condition is a true hypertrophy, the larger the tumor will be. On the contrary, visible peristaltic waves as well as a palpable tumor mass are seen, even following operation, though clinical symptoms are not present. These patients as well as those not operated on, when treated medically, lose these physical manifestations. At necropsy, in the patients who had been operated on and who had died several weeks or months later of some other disease, there has frequently been found a persistence of the pyloric hypertrophy and in addition a marked hypertrophy of the wall of the stomach. In one of the author's patients who died five days after operation, there was scarcely any trace of an incision. The author raises the question as to what extent the nervous mechanism is interfered with when a Ramstedt incision is made.

He tried to prove this experimentally by applying a 10 per cent solution of barium chloride to the pylorus. He actually produced a pylorospasm which he treated surgically by the Weber-Ramstedt incision and found that in five days such an incision had entirely healed and that the only thing noted was simply a vigorous peristalsis. To prove the thoroughness of the healing of the muscle, the author reapplied barium chloride to the stomach proximal to the ring to institute some really vigorous peristaltic waves and noticed that the muscle fibers close to the incision did not participate in the contraction. He ascribed this to the destruction of their nerve elements.

Owing to the early closure of the Ramstedt incision in the pyloric ring, Hildebrand modified this operation by cutting out a wedge-shaped section of the pylorus. He did this in six children with excellent results, and now this method has become the operation of choice, especially in cases in which the longitudinal muscle fibers are especially preponderant.

The author discusses the nature of congenital pyloric stenosis — whether it is a hypertrophy of pyloric muscle, a primary congenital anomaly, or whether it is acquired due to a pyloric spasm. The opinion of some is that it is a pylorospasm in an already congenitally hypertrophied muscle.

In reviewing the literature of late results of operations for pyloric stenosis, he calls attention to Heubner's fifteen patients. Two of this series had disturbances of the stomach in that they could not tolerate solid foods until the third or fourth year. In Hildebrand's clinic, twenty patients were operated on and described by the author. Of this series, only two had digestive disturbances.

Concerning pylorospasm in neuropathic children, Heubner found four out of fifteen with outstanding nervous disturbances consisting of nervousness, restlessness, cramps and nocturnal enuresis. Liefmann could establish nervous symptoms in 50 per cent, while Lichtenstein could establish them in 80 per cent, and, finally, the author, in 25 per cent. The influence of heredity has been traced by some investigators in as high as 52 per cent of the cases, but in the author's own series it was only 20 per cent.

THE SURGICAL RESULTS OF TUMORS OF THE KIDNEYS IN CHILDHOOD. HANS WARNER, Arch. f. klin. Chir. **145**:347, 1927.

Tumors of the kidneys in children are generally of the mixed type, containing muscle, cartilage and sometimes epithelium. In adults the common tumors found in the kidneys are hypernephromas and carcinomas, and 95 per cent of all tumors of the kidneys occur in adults. Of all tumors in children 20 per cent occur in the kidney, and of all tumors in adults only 5 per cent occur in the kidney.

The symptoms common in adults are missing in children, since hematuria is present in only about from 10 to 15 per cent of children, a palpable tumor mass in 80 per cent and pain in only from 25 to 30 per cent. The mortality in adults in all operative cases for tumor of the kidneys is 70 per cent and in children, from 80 to 90 per cent.

The author feels that the only therapy is surgical intervention, and that for this to be successful, early diagnosis is most essential. To facilitate diagnosis, he emphasizes six essentials; namely, location, size, contour, consistency, motility and pain. The kidney in children lies deeper than in adults and tends to fill the iliac fossa much earlier. Tumors of the kidney must be differentiated from those of the spleen, liver and bowel. Pain is a symptom occurring late and usually indicating a rapidly growing tumor. Later there are associated vomiting, obstipation, diarrhea, and distention of the superficial veins of the abdomen. Hematuria may occur early or late, and temperature is usually of a most atypical type. Finally, emaciation, anemia and cachexia develop. In addition to all these symptoms, the urologic examination is an important aid in determining any congenital anomalies and the condition of the opposite kidney. In addition, the author emphasizes the use of cystoscopy, pyelograms and renal functional tests, since the urine may not show pathologic changes at any time.

The author recommends two types of incisions: transperitoneal and extraperitoneal. Sometimes a combination of both is used, depending usually on the location and size of the tumor. The author regards the extraperitoneal lumbar incision as the incision of choice, since it may be enlarged to become a transperitoneal incision.

The immediate mortality is from 70 to 90 per cent. Of all recurrences, 75 per cent occur in the first year. In Hildebrand's clinic, the recurrence was 50 per cent during the first year, and most of the patients operated on were brought in in advanced stages of the disease.

Of great importance are the postoperative and subsequent management. The use of the roentgen ray is advised, and the resistance of the children should be kept high, for often, being in a weakened condition, they succumb to some intercurrent disease.

OPERATIVE TREATMENT OF HYPOSPADIUS IN THE MALE. WERNER BIRKENFIELD, Arch. f. klin. Chir. **145**:445, 1927.

The operative treatment of hypospadius is a problem still to be solved for all forms of the condition. The operative problem is determined according to the type and severity of the anomaly.

The mildest form, hypospadia glandis, is one in which the external urinary meatus is on the undersurface of the glans at the level of the sulcus coronarius, causing little if any functional disturbance. This physical defect should be corrected for psychic reasons. The operation of choice for this form is the method of Beck-von Hacker-Bardenheuer and consists of lengthening the urethra by cutting around it and fixing it to the tip of the glans penis. The ideal time is between the ages of 2 and 4 years.

For the moderately severe forms such as hypospadia penis, or penoscrotal hypospadia, the most common form with acute bending down and retraction producing considerable functional disturbance, the operative method is varied. If the opening is near the glans, the method employed in the mild form may be used. In the more severe forms, and these should be taken care of at least by the third month, Duplay's method is the one of choice, although two other types of operation are really applicable. Duplay's method consists of making two raw surfaces and suturing them together over a catheter. In a second method, the skin of the shaft is used to construct a new canal; this is known as the Thiersch method. It consists in forming two flaps along the urethral groove, one being used to build a canal and the other for the outer covering. This method is not successful if the skin is limited, although scrotal

skin and skin transplants have been used, but these tissues are not accustomed to urine and therefore are easily irritated and inflamed. If the foreskin cannot be used, the result will be unsatisfactory.

The group presenting the greatest problem is the hypospadia scrotalis and perinealis with which a small penis and, sometimes, bilateral undescended testes usually are associated and may be so severe as to make determination of sex difficult. In these conditions there is usually incontinence of urine, and for this group the author recommends the method of Thiersch.

The author reports four cases of hypospadius and one epispadius, all were operated, and in four there developed a small urinary fistula, all of which were closed by a minor secondary closure.

SURGICAL TREATMENT OF ATRESIA ANI CONGENITA. ARTHUR KUTTNER, Beitr. z. klin. Chir. **138**:757, 1927.

The author discusses several classifications. One advanced by von Esmarch, many years ago, has been replaced by a classification made in Bier-Braum-Kummel's surgery and consists of the following:

I. Atresias without fistula.
   1. Atresia ani simplex.
   2. Atresia recti.
   3. Atresia ani and recti.

II. Atresias with internal fistuli.
   1. Atresia ani vesicalis et urethralis.
   2. Atresia ani vaginalis.
   3. Atresia ani uterina.

III. Atresias with external fistuli.
   1. Atresia ani perinealis.
   2. Atresia ani scrotalis.
   3. Atresia ani suburethralis.
   4. Atresia ani vestibularis.

The author chooses to omit groups I and III and to discuss only atresias with internal fistuli, and of this group, especially, atresia ani vesicalis and urethralis. In this condition, there is complete absence of the anal portion with a rather high closure of the large bowel. The communication is most often found at the fundus of the bladder, occasionally at the neck of the bladder and sometimes at the prostatic urethra. The surgical treatment of these types is beset with many difficulties, usually requiring two stages, and is associated with a long convalescence and occasionally with infection and sloughing of tissue.

In bladder-rectal fistulas, the method of Hochenegg and that of Stettner are suitable. They likewise require two stages: one consisting of a laparotomy in which the communication is divided, both bladder and rectum closed and the lower bowel mobilized and the other stage, in which it is planted into the perineum.

In atresia ani urethralis, Stettner employs a two-stage operation. The first stage consists of the formation of a bed for the bowel opening with exposure of the communication. The second stage, performed from fourteen to forty days later, consists of a division of the communication and the implantation of the lower bowel. This method may also be used to good advantage when the communication is with the lower part of the urinary bladder.

The author emphasizes the fact that there is no uniformity of opinion concerning operative methods, but that in most all bladder-rectal defects a two-stage operation is necessary. The author reports one of his own cases, that of a new-born child, in which three operations were necessary before the condition was corrected. The first stage consisted of a proctoplasty, the second of a preternatural anus, and in the third, a flap was made for closing both openings.

OBERHELMAN, Chicago.

## Miscellaneous

THE RELATIONSHIP OF CERTAIN FOCAL INFECTIONS TO GENERAL DISEASE IN INFANTS AND YOUNG CHILDREN. McKIM MARRIOTT, Ann. Int. Med. **1**:1 (July) 1927.

Dr. Marriott summarizes the evidence for the relationship of focal infections of the mastoid antrums and paranasal sinuses to diarrhea, vomiting and failure to gain weight in infants, and to nephritis, heart disease, chorea, malnutrition, asthma, and cyclic vomiting in older children. He outlines the treatment of such patients by surgical drainage of the infected sinus.

GIBSON, Elkhart, Ind.

METHYL SALICYLATE POISONING: REPORT OF A CASE. D. P. ARNOLD and A. W. JACOBSEN, Bull. Buffalo Gen. Hosp. **5**:44 (Dec.) 1927.

A fatal case of methyl salicylate poisoning in an infant, aged 10 months, is presented, this being the seventh case reported. The outstanding clinical signs were stupor with markedly increased reflex excitability and an extreme degree of hyperpnea. The respirations were deep, like those in patients with severe acidosis, but at the same time were rapid. This type of respiration together with the odor of acetone on the breath and the presence of large quantities of ketone bodies in the urine would lead one to suspect the presence of a ketone body acidosis.

The cause of death is probably circulatory collapse, of either the vasomotor or respiratory centers. There is usually a severe renal damage, acute edema of the lungs, degeneration of the heart and fatty degeneration of the liver.

SANFORD, Chicago.

TRANSVERSE LINES IN X-RAY PLATES OF THE LONG BONES OF CHILDREN. MARTHA M. ELIOT, SUSAN P. SOUTHER and E. A. PARK, Bull. Johns Hopkins Hosp. **41**:364, 1927.

The transverse lines seen so frequently in roentgenograms of children and the anatomic conditions responsible for their appearance are described. The lines are formed by the enlargement and branching of the bone trabeculae forming an irregular latticework perpendicular to the main axis of the bone. The formation has its origin at the junction of cartilage and shaft. Owing to the retardation of growth, the bone building energy of the shaft concentrates at a single level, resulting in the formation of trabeculae which extend transversely.

The dense line at the end of a healing rachitic bone is produced by a sheet of calcium phosphate. It owes its visibility to the fact that the rachitic zone between it and the end of the shaft is almost free from lime salts, thus allowing the provisional zone to show by contrast. When rachitic lesions heal completely, the former diseased area in the bone never shows a line as a residuum in the roentgenogram, as is often supposed, but always manifests itself as a transverse band of increased density. This band represents the portion of the bone formed in the rachitic process and is always a transverse disk of from several millimeters to 2 cm. in thickness.

LYTTLE, New York.

THE PRESENT STATUS OF MATERNAL AND INFANT HYGIENE IN THE UNITED STATES. L. K. FRANKEL, Metropolitan Life Insurance Co., 1927.

Over the period of 1918 to 1925 in the United States birth registration areas, there was a decline of infant mortality of between 4 and 5 per cent a year. The infant death rate declined more rapidly in urban than in rural areas, and slightly more rapidly for female than for male infants. Beyond 1 month

of age, there was a fall of 41 per cent in the infant death rate, or an average of 7.4 per cent per annum. Under 1 month of age, the death rate declined only 15 per cent or on an average of 1.8 per cent a year.

The death rate from prematurity shows a decline of 0.8 per cent a year. The death rate from congenital malformations declined about 0.6 per cent a year.

The most startling fact was the sharp increase during the past ten years in the death rate from injuries at birth. This increased more than 5 per cent a year. Injuries at birth made up 14.5 per cent of the deaths on the first day of life in 1925, and for 16.7 per cent of the deaths in the first week, excluding the first day.

SANFORD, Chicago.

AIDS IN MANAGEMENT OF BREAST MILK STATIONS. MARIE-ELISE GAYSER, München. med. Wchnschr. **50**:2142, 1927.

Three points are considered: first, methods of distinguishing between human and cow's milk; second, use of soured human milk, and third, use of dried human milk.

It is necessary to distinguish between human and cow's or goat's milk to avoid fraud. The author believes that this can be done by examining the milk in direct sunlight. Human milk has a violet or blue fluorescence, while cow's or goat's milk has a yellow fluorescence. Five cubic centimeters of a sample will suffice. Two known samples should be used as controls.

Human milk that has soured need not be thrown away, but may be fed to children already on acidulated milk.

Dried human milk powder works satisfactorily, but at present its production is too expensive a process.

SCHORER, Kansas City, Mo.

FATIGUE IN CHILDREN. GEORGE R. IRVING, New York State J. Med. **27**:764 (July 15) 1927.

Fatigue may be merely a symptom, occasionally a disease process in itself or an abnormal process which predisposes the child to more serious disorders.

Effort beyond the child's ability and lack of balance between the mental and physical capacity head the list as causes of fatigue, while constant association with adults and the attempt to keep up with older children follow.

Overwork in school, studies outside the school, overplanned routine, daylight saving, unsupervised athletics, too heavy clothing, eye strain, poor ventilation, overeating, nasal obstruction, worry and constant "nagging" are frequent causes of the modern picture of fatigue in children.

AIKMAN, Rochester, N. Y.

ULTRAVIOLET LIGHT TREATMENT. ETHEL CASSIE, Lancet **213**:1121 (Nov. 26) 1927.

The result of a year's work at the Carnegie Infant Welfare Institute is given. The treatments were given twice weekly, and a course consisted of twelve treatments. From one to two courses sufficed to produce a normal roentgenogram in the large majority of cases of rickets. Encouraging results were obtained in "debility following the infectious diseases" and in catarrhal children. Infants with eczema under this treatment did well. Wasted infants suffering from chronic dyspepsia from various causes did not benefit to any marked extent.

LANGMANN, New York.

DISEASES OF CHILDREN IN 1927. ANNUAL REVIEW. P. LEREBOULLET and F. SAINT GIRONS, Paris méd. **45**:340 (Nov. 5) 1927.

A complete and comprehensive résumé of the progress of French pediatrics during the last year is given. A few of the more important features are as follows:

In the diseases of the new-born, pulmonary conditions have received much attention. Many investigators believe that a great number of infections of the lungs are due to a hematogenous infection that localizes in the lungs. Attention is called to the studies of Debré, Semelaigne and Cournand on infectious edema of the lung. They believe that the pneumococcus is the causative organism.

There is a tendency toward the use of dried milk in infant feeding and considerable success has been obtained by the employment of various modifications of acid milk. Nobécourt's feedings of large quantities of carbohydrate to cachectic infants, who, he claims, have a high tolerance, has resulted in the feeding of more sugar; the amount given is about 30 Gm. per kilogram of body weight. Lesné has shown that many sick infants are underfed, and he believes that diarrhea and vomiting are often caused by underfeeding. Athreptic infants should receive 250 calories per kilogram of body weight.

In the chronic conditions, the greatest strides have been made in infantile tuberculosis, in which Calmette's B.C.G. vaccine has resulted in a great reduction of infant mortality from tuberculosis. Syphilis has also decreased by systematic prenatal treatment of the mother.

In other conditions, infants with eczema have been treated satisfactorily both with an autogenous vaccine and with small doses of insulin. A stock mixed vaccine has been giving some satisfactory results in asthma. The close connection between asthma and tuberculosis has also been under considerable investigation. The value of a mixed vaccine has been demonstrated in many cases of bronchopneumonia.

SANFORD, Chicago.

SOME SPECIAL CONDITIONS OF TOLERANCE TO STRYCHNINE MEDICATION IN PEDIATRICS. BRUNO TRAMBUSTI, Riv. di clin. pediat. **25**:669, 1927.

A child, aged 25 months and weighing 25 Kg., was being treated for diphtheritic paralysis by injections of strychnine sulphate. Through a mistake of the apothecary, 1/15 grain (0.004 Gm.) was given instead of the 1/60 grain (1 mg.) ordered. The child soon began to have convulsions in the unparalyzed muscles. Convulsive movements persisted for four hours, and some symptoms persisted for twenty-four hours. The child recovered satisfactorily from her paralysis.

The dose of strychnine given is estimated by the author to be lethal to normal children of 25 months weighing 12 Kg. Therefore the conclusion is reached that a child suffering from diphtheritic paralysis can tolerate greater doses of strychnine than a normal child.

HIGGINS, Boston.

REPORT OF MUNICIPAL HEALTH UNITS FOR THE FISCAL YEAR ENDING JUNE 30, 1927. Porto Rico Rev. Public Health and Tropical Med. **3**:102 (Sept.) 1927.

The Porto Rican local health departments functioning through the sanitary police, whose duties are sanitary policing and suppression of epidemics, was found inadequate. The new organization is to provide public health education, control of communicable diseases including hook worm disease and tuberculosis, and child hygiene with medical inspection of schools. These goals are being rapidly attained within the first year of operation.

BARNETT, Chicago.

THE OPERABILITY OF NURSING INFANTS. E. HAGENBACH, Arch. f. klin. Chir. **145**:635, 1927.

The question of operation in nursing infants is a debate that often takes place between the surgeon and the pediatrician. The question, however, usually resolves itself into one in which either the danger of operation outweighs the

benefit or vice versa, and the answer is simply a good reason for early operation. The conditions for which operation should be performed constitute two large groups — one, emergency and the other, nonemergency. In the latter group such conditions as hernia, orthopedic conditions, harelip and cleft palate are found. One advantage of early operation in these nonemergency conditions is that a small child, other things being equal, is much more easily handled.

The mortality in a series of eighty-two nursing infants, none over 10 weeks old, who were operated on, consisted of only five deaths: one from atresia ani operation; another from strangulated umbilical hernia; a third from a meningocele; a fourth from aspiration pneumonia following herniotomy, and a fifth from resection of an ectopia vesicae. The conditions for which this series of eighty-two infants were operated on were nonemergency, the operations having been done at a time most ideal for such conditions. The author feels that in these conditions which, sooner or later will require surgical intervention operation should be performed, and the sooner the better.

OBERHELMAN, Chicago.

CONCERNING HOSPITALISM. E. OBERWARTH and LANGE, Gesundheit. f. d. Kindesalter **1**:427 (May) 1926.

The article represents a criticism of the influence of hospitalism in the City Home for Children and Mothers at Charlottenburg, West-End. The authors ascribe the small number of infections to the use of the mask for mothers and nurses.

For the home, the number of deaths to every 10,000 nursing days was 2.8 per cent for 1924; for 1921, the figure was 5.1. In five years only, about 1.7 per cent of the infections occurring in the home resulted in death. Although the body weight and height almost approximated the Pirquet and Camerer figures, the children generally were late in learning to sit and walk. This could not be due to rickets. The authors therefore ascribe it to the lack of stimulation from the surroundings of the child. The monotony of the infant's existence in such a home often leads to stereoptic movements.

THE "HIGHER MORTALITY" OF ILLEGITIMATE INFANTS IN BERLIN. CLEMENS WIESE, Gesundheit. f. d. Kindesalter **1**:439 (May) 1926.

The mortality of illegitimate infants due to prematurity is higher; perhaps the occurrence of prematurity is more frequent. Syphilis is more frequent. In the first four to six months these infants are more endangered by nutritional disturbances, due to the frequent failure of breast feeding and the frequent change of foster parents. The illegitimate infant seems to be more endangered than the legitimate by common respiratory infections in contagious diseases, due, perhaps, to the fact that he is more often given institutional care.

REICHLE, Cleveland.

# Society Transactions

## CENTRAL STATES PEDIATRIC SOCIETY

*Twelfth Annual Meeting, Cleveland, Oct. 14-15, 1927*

O. L. Goehle, M.D., *President, in the Chair*

*Program for Friday Morning Session, Amphitheatre, Babies' and Children's Hospital, Oct. 14, 1927:*

Demonstration of Roentgenograms and Photographs from the Babies' and Children's Hospital. Prepared by Mr. David Shields and shown by Drs. Nourse, Hartman and Gerstenberger.

Organization and Method of Operation of Institutional Care of Children in Cleveland. Dr. S. A. Wahl.

Syphilitic Tumor of the Stomach in a Twelve Year Old Girl. Dr. W. M. Champion.

Clinical Observations with Injections of Dextrose in Diphtheria. Dr. J. A. Toomey.

Demonstration of Pathologic Specimens. Dr. H. S. Reichle.

Intrascalp Method of Administration of Neoarsphenamine. Dr. L. R. Brigman.

A Case of Congenital Heart Block. Dr. J. H. Davis.

A Case of Myelogenous Leukemia. Dr. W. B. Taggart.

Subacute Bacterial Endocarditis. Dr. J. D. Nourse.

Hemihypertrophy with Macrodactyly, Multiple Nevus and Other Malformations. Dr. C. T. J. Dodge.

Epidermolysis Bullosa Hereditaria. Dr. C. T. J. Dodge.

Nevus Pigmentosus Pilosus. Dr. C. L. Ruggles.

A Cod Liver Oil Requirement Curve. Dr. N. C. Wetzel.

Data Concerning the Dose of the Antirachitic Factor in the Prevention and Cure of Rickets. Dr. H. J. Gerstenberger.

Rachitic Spasmophilia. Dr. J. I. Hartman.

Heart Block in Diphtheria with Recovery. Dr. Elizabeth Bartos.

Allergic Manifestations in Tuberculosis. Dr. C. W. Burhans.

Congenital Anemia. Dr. J. E. McClelland.

Foreign Body in the Bronchus of a Child with Congenital Absence of the Other Lung. Dr. O. L. Goehle.

Inspection of New Babies' and Children's Hospital.

### Demonstration of Roentgenograms and Photographs from the Babies' and Children's Hospital. Prepared by Mr. David Shields and shown by Drs. Nourse, Hartman and Gerstenberger.

Dr. Nourse: During the last few years, the diseases described by Perthes, Köhler, Osgood, Schlatter, Scheurman and others have been generally considered particular manifestations of the general condition, osteochondritis juvenilis deformans or at least of similar processes affecting different epiphyses. Occasionally a case presenting two or more of these lesions has been reported.

The case to be demonstrated exhibits the following lesions: Perthe's disease, bilateral; vertebral osteochondritis; Köhler's disease of the metatarsal bones, fourth, bilateral; similar change in the metacarpals, fifth bilateral; osteochondritis of the head of the left humerus, the right, questionable; osteochondritis, distal end, ulna, bilateral; change in pisiform bones, bilateral; delayed development of carpal scaphoid, bilateral; olecranon apophysitis, bilateral; epiphysitis, lower end of humerus, bilateral; apophysitis of tibial tuberosity, bilateral; changes in some of the teeth; change in the head of the first metatarsal bone, bilateral.

Dr. Hartman showed a series of lantern slides demonstrating: (*a*) Active nonhealing rickets; (*b*) healing rickets in various stages as evidenced both at the ends of the long bones and at the periosteum.

Dr. Gerstenberger showed lantern slides illustrative of the following: (*a*) scurvy at the distal ends of the radius and ulna and at the distal end of the femur; (*b*) pulmonary tuberculosis — the miliary type and the peribronchial type of a widely distributed character often erroneously accepted as being characteristic of miliary pulmonary tuberculosis.

Organization and Method of Operation of Institutional Care of Children in Cleveland. Dr. S. A. Wahl.

For the past four years, a special pediatric clinic has been in operation at the Lakeside Dispensary. This clinic was organized for two main reasons: first, to give ready physical examinations on admission to children prior to placement in either boarding homes or institutions; second, to separate this type of service from the regular pediatric division so that the clinicians would not be interrupted in their pursuit of clinical disease entities.

Such an arrangement has provided for the following:

1. Preventive measures against contagious disease.
2. Diagnostic measures as to syphilis, tuberculosis, malnutrition and chronic diseases in general.
3. Consultation service with the dispensary departments on other than pediatric conditions.
4. Follow-up in the twenty-three institutions that care for children with the provision for all forms of medical and dental services.
5. Proper linking of the social and medical problems involved, much to the advantage of the welfare of Cleveland's underprivileged children.
6. Complete mental and behavior study of each child at the Child Guidance Clinic.

A study was made referable to the medical status of the cases. It was found that 95 per cent of all the children had one or more defects, the average being 2.5 per child. The distribution of the defects in 6,000 children are shown in the accompanying table.

*Distribution of Defects in Six Thousand Children*

| | Per Cent |
|---|---|
| Dental caries | 55 |
| Simple goiter | 53 |
| Malnutrition | 23 |
| Ocular disturbance | 19 |
| Obvious nervous disease | 5 |
| Diseased tonsils | 13 |
| Disease or defect of the genitalia | 15 |
| Other defects, such as the acute respiratory infections, skin diseases, contagious disease, etc. | 64 |

The clinic has served to raise the medical standards of the institutions in which children are cared for and is aiding in the solution of the social problems of the on-coming generations by eliminating two causative factors, physical defect and disease.

Syphilitic Tumor of the Stomach in a Twelve Year Old Girl. Dr. W. M. Champion.

Irene, B., aged 12 years, complained of vomiting, pain in the abdomen, and failure to grow and gain weight properly. These symptoms had been noted for about a year and had become aggravated within the last three weeks. Pain was intense after meals and was relieved by vomiting. Even water was returned, and vomiting occurred regularly a half hour after eating. The girl's appetite was poor; she had not grown in height for some time and was losing weight

rapidly. She had frequent headaches, and was constipated. At times, there were streaks of blood in the stools.

The parents vigorously denied venereal disease and all symptoms suggestive of such disease.

The girl had had pneumonia twice during the first year of life and again at the age of 3. She had osteomyelitis of the right knee at 6 years but had recovered fully.

The girl was somewhat stunted and was easily 20 per cent underweight, with a pale skin and a dull expression. She had definite Hutchinson teeth, a slight but definite enlargement of the thyroid and some tenderness in the region of the umbilicus. It was thought that the liver and spleen were slightly enlarged.

In the hospital, it was found that on several different occasions the blood Wassermann reaction was definitely positive. The Wassermann reaction with the spinal fluid was positive, and six cells were present in the fluid. There was a moderate degree of anemia. The roentgenogram of the stomach after a barium meal showed a marked filling defect of the entire pylorus, especially along the greater curvature, and evident thickening of the wall of the pylorus. There was slight delay in emptying time.

During a month of residence in the hospital, the symptoms as detailed by the mother on the first visit were confirmed. The diagnosis was made of congenital syphilis with a syphilitic tumor of the stomach. Antisyphilitic treatment consisting of potassium iodide and arsphenamine was instituted. It was hoped that the result of the treatment would further confirm the diagnosis.

Shortly after treatment was instituted the vomiting ceased, the appetite returned, the pain disappeared, and the girl began to gain in weight. Her weight increased from 50 pounds (22.7 Kg.) on admission to the hospital to 69 pounds (31.3 Kg.) in six months. She now has no symptoms. Subsequent roentgenograms of the stomach show definite improvement, but the tumor mass is still visible, as manifested by a persistent filling defect. Laboratory or physical evidence of syphilis is not present in any other member of the family.

CLINICAL OBSERVATIONS WITH INJECTIONS OF DEXTROSE IN DIPHTHERIA. DR. J. A. TOOMEY.

After injections of dextrose, the systolic blood pressure rises from a few to twenty points and the diastolic from a few to as many as forty points. The rise in diastolic blood pressure is appreciated almost immediately after injection.

The collapsed veins, which frequently are present, become filled again; the general cyanosis of the patient becomes modified in a short time; the cheek loses its pasty color and the skin, its clammy feel; a healthy sweat breaks out, and adults often say that they feel better.

The number of heart blocks and cases of acute cardiac dilatation occurring after admission to the hospital have decreased. When heart block occurred, it was possible to prolong life in one instance for fourteen days, in another for five days, in another for eight, and in another for nine days. No patient, however, has lived. Of forty-six patients with "collars of glands," twenty-four have lived, while in previous years all died.

The amount usually injected is from 50 to 75 cc. of a 20 per cent solution of dextrose. In severe forms of diphtheria, this is done twice or as often as four or five times a day, when the diastolic pressure falls below 40 mm. of mercury. The injection is made slowly, over a period of from ten to fifteen minutes, into any accessible vein. Sugar is not found in the urine, although from 30 to 50 Gm. may be given intravenously in a short time.

The mechanism bringing about the improvement may be either hydrostatic per se, hydrostatic with secondary osmosis, metabolic, or a combination of any one of these.

From a clinical experience of three years, I believe that certain types of diphtheritic patients react well to intravenous injections of dextrose.

DEMONSTRATION OF PATHOLOGIC SPECIMENS. DR. H. S. REICHLE.

Serial roentgenograms were shown of three patients with pulmonary tuberculosis and one with postpneumonic multiple abscess formation with pyothorax. With the aid of the material obtained at autopsy the attempt was made to show how far the roentgenograms described the true pathologic picture. In the case of a complete consolidation of the middle lobe, it was shown that in the roentgenogram a clear area between the diaphragm and the lower border of the shadow corresponded to the normal right lower lobe (fig. 1).

Fig. 1.—Clear area between the diaphragm and the lower border of the shadow corresponding to the normal right lower lobe.

INTRASCALP METHOD OF ADMINISTRATION OF NEOARSPHENAMINE. DR. L. R. BRIGMAN.

The treatment of patients with syphilis is an old story, so it will conserve time to outline only the routine used at the Babies' and Children's Dispensary, in the intrascalp administration of the arsenicals.

The intrascalp injection of neoarsphenamine or sulpharsphenamine is the method of choice in infants with congenital syphilis who are under 18 months of age, because it embraces all the desirable features of every other method and at the same time eliminates all the objectionable points. The intrascalp injections are quick, easy, simple and safe, and are comparatively painless and efficacious as compared with the intrasinus, intravenous, intramuscular, intraperitoneal and rectal routes.

The technic consists of washing the parietal area of the scalp with 95 per cent alcohol, painting with tincture of iodine, and washing again with alcohol. A 25 gage needle, 1½ inches long, is inserted at a point 1½ inches above and behind the ear, the point of the needle being directed downward and posteriorly

to the mastoid. After the solution is injected slowly, the resulting mass is massaged to disperse it through the surrounding tissues, and collodion is applied to the needle puncture. Absorption starts immediately and is complete in a few hours.

This procedure is easier than intravenous injections and eliminates the possibility of reactions to the drug. It is less painful than the intramuscular injection into the gluteal muscles and does not leave any nodules.

The solution used must consist of 0.03 Gm. of neoarsphenamine or sulpharsphenamine in each cubic centimeter of sterile water; not more than 4 cc. of this dilution must be put in the parietal area at one time, or swelling of the surrounding tissue results. However, both parietal areas may be used at one sitting to get the proper dose for the body weight of the patient.

This method has been used more than a thousand times without a reaction from the drug, an abscess from the technic, or a slough of the tissue. It has also taught me that infants with congenital syphilis do not have large veins in the scalp unless they are pale and underweight from any cause whatever. Another interesting fact is that the scalps affected by congenital syphilis that I have seen are rarely ever covered with a wig.

Curative results are as readily obtained by this method as by any other.

A Case of Congenital Heart Block. Dr. J. H. Davis.

A boy, aged 22 months, was first seen in January, 1927, at the age of 13 months, when he was admitted to the pediatric service of the City Hospital as a "boarder." At the examination it was noted that the child had a marked bradycardia, the pulse and apical rate approximating 40-44. He also had a loud systolic blow which was heard best at the apex, but was also heard readily throughout the entire chest. Cyanosis, clubbing of the fingers, and a history of syncope or convulsions were not present.

An electrocardiographic tracing showed a complete heart block which, because of the age of the patient and the lack of history of acute attacks, was assumed to be congenital. The ventricular rate was 45, the auricular rate was 125. After the administration of atropine in physiologic dosage, the ventricular rate remained at 44, while the auricular rate went up to 150. During the course of an attack of bronchopneumonia the ventricular rate reached 56, while the auricular rate went up to 156. In September a new tracing was made, and the block is still present. In the meantime the child has been growing slowly and has been free of any symptoms referable to the cardiac defect.

Sixteen cases of congenital heart block have been previously reported in the literature, all with graphic proof of the block. In only one, the most recently reported case, that of Wilson and Grant, were serial studies of the conduction system made. This showed an interruption in the bundle associated with a defect in the interventricular septum such as is presumed to exist in most of these cases.

A Case of Myelogenous Leukemia. Dr. W. B. Taggart.

The particular reasons for presenting this case of chronic myelogenous leukemia in a boy, now aged 10½ years, are: (1) Duration of the condition and (2) change in the condition following roentgen-ray therapy and splenectomy.

The patient was admitted to the hospital in March, 1926, with complaints of weakness, malnutrition, pallor, tachycardia, shortness of breath on exertion and poor appetite, all of which dated from about three months before admission. The boy was undernourished and pale and had generalized adenopathy and an enormous spleen which filled the left half of the abdomen and extended to a point midway between the umbilicus and the symphysis. Examination of the blood showed: normal bleeding and clotting time; 208,000 white blood cells: 65 per cent of which were myelocytes; 45 per cent hemoglobin, and 2,500,000 red blood cells.

Splenectomy was advised and the child was transferred to the Lakeside Hospital, where the spleen was first subjected to roentgen-ray therapy and reduced one half in size and then removed. Almost immediately following the operation, improvement in the child's general condition appeared and, except for the interruption which I shall again refer to, has remained excellent.

He was again admitted to the hospital after seven months with an acute attack of priapism (fig. 2) and fever, with evidences of nasopharyngeal infection, signs of which had been evident for ten days before he came to the hospital. With palliative treatment, sedatives and repeated deep roentgen-ray therapy over the long bones, this condition subsided.

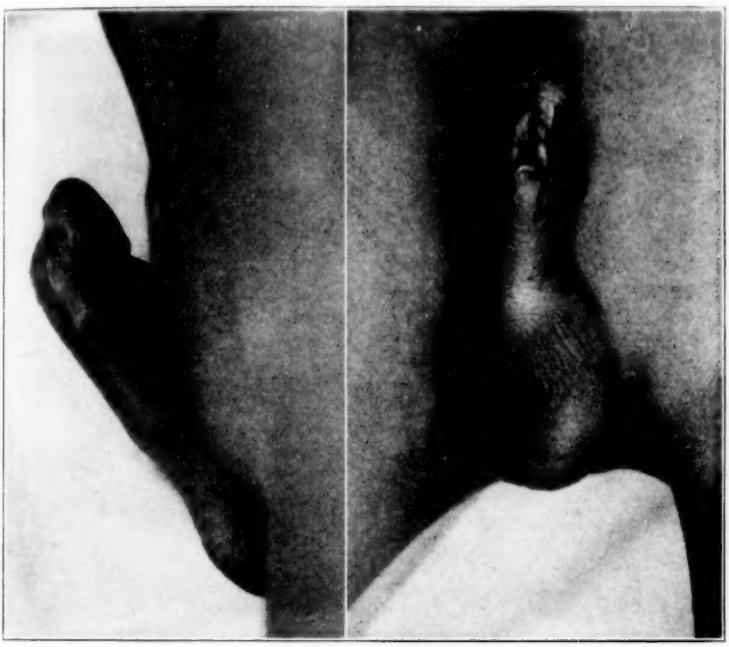


Fig. 2.—Priapism in a patient in whom splenectomy had been performed.

The patient responded well to roentgen-ray therapy and splenectomy. His white blood cell count fell following every roentgen-ray treatment, which was given after the count had risen to 150,000. After this drop the count again led to a high level.

The differential determinations at the present time show a slightly lower percentage of abnormal white cells.

The patient's weight during the eighteen months after he first came under observation increased 4 pounds (1.8 Kg.). His appetite is good. He attends school regularly and is in good spirits.

SUBACUTE BACTERIAL ENDOCARDITIS. DR. J. D. NOURSE.

Four cases of subacute bacterial endocarditis due to *Streptococcus viridans* were reported in children, aged 10 years or younger. The duration of the disease was from two to nine months. All the cases ended fatally. Two of the children had suffered previous cardiac damage, one a congenital heart lesion and the

other rheumatic valvular disease. All presented the four criteria for a positive diagnosis: continued fever, evidence of valvular disease, embolic phenomena and a positive blood culture for *Streptococcus viridans*.

Another case was reported in a child, aged 2½ years, who showed a streptococcic septicemia. This patient apparently recovered following an intravenous course of sodium cacodylate, but died later from an acute infection of the upper respiratory tract. At autopsy, an old interstitial pneumonia, pleuritis and adherent pericarditis were found.

A sixth case was in an infant who was 5 weeks of age on admission and had been in the Babies' and Children's Hospital for five and one-half months. During this time an intermittent fever varying from 38 to 40 C. (100.4 to 104 F.) daily had been present, notwithstanding a steady gain in weight. *Streptococcus viridans* had been been isolated from the blood stream. Evidence of valvular disease was not present. One roentgenogram of the mastoids was regarded as suspicious. One taken previously was reported negative. Other foci of infection have not been discovered.

DISCUSSION

QUESTION: What media are you using?

DR. NOURSE: The blood is taken into dextrose broth and into melted dextrose agar, plates being poured from the latter.

HEMIHYPERTROPHY WITH MACRODACTYLY, MULTIPLE NEVUS AND OTHER MALFORMATIONS. DR. C. T. J. DODGE.

Congenital hypertrophy may be partial, crossed or unilateral. The unilateral type, or hemihypertrophy, is uncommon but is not so rare as reports in the

Fig. 3.—Varicose veins in a patient with hypertrophy.

literature would lead one to believe. Like the nevi with which it is so frequently associated, it may be noticed shortly after birth, rather than at birth; nevertheless, it is congenital. A hereditary factor has not been discovered as yet, but the occasional association with polydactyly leads one to believe that there may be an obscure hereditary factor present. The disparity usually increases during the period of rapid growth but may decrease in adolescence.

Many interesting anomalies may be associated. The skin may show an increased vascularity, causing a reddening or cyanosis of the part involved, or it may be mottled, forming the so-called cutis marmorata. Port wine nevi, nevus unius lateralis, telangiectasis and pigmentations are frequently found. On the hypertrophied side, the epidermis may be rough or may show increased desquamation. Unilateral hyperhidrosis and hypertrichosis may be present. The nails may be coarse and rigid, and the teeth may erupt earlier on the precocious side. Varicose veins are not infrequent. Tumors such as lipomas, fibromas, neurofibromas and, more rarely, chondromas, have been associated with various cases. Of deformities of the bones one may find polydactyly, syndactyly, osteophytes, and ankylosis, genuvalgum, talipes valgus and scoliosis. The paired organs may be enlarged on the hypertrophied side. The brain and the eyeball may show enlargement. Mental deficiency is present in about 15 per cent of the cases so far described.

The case that is demonstrated is about the most complete museum of hemihypertrophy and associated anomalies which has so far been described. The patient is a boy, aged 5 years. The right side is enlarged. The skin on the right side is coarse and dry. Extensive port wine nevi are present on the right side and left leg. Large varicose veins are to be seen in both legs (fig. 3). A nevus unius lateralis is present on the right arm. Numerous lipomas and fibromas are present, and elephantiasis-like pads cover the palmar surfaces of both hands and all the fingers. The lower central incisor is a permanent tooth and was erupted when the patient was about 3 years old. A bony ridge occupies the sagital line in the vertical portion of the skull. Scoliosis, genu valgum and macrodactyly are present. The mental condition is that of a child aged about 1 or 1½ years.

EPIDERMOLYSIS BULLOSA HEREDITARIA. DR. C. T. J. DODGE.

There are three salient points in the diagnosis of this peculiar and rare anomaly of the skin:

1. It is characterized by the development of vesicles and bullae on even slight traumatic provocation. The contents of these are serous, more rarely serosanguineous and seropurulent only when the infection is secondary.

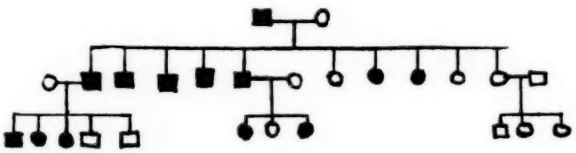


Fig. 4.—Incomplete chart, showing persons affected with epidermolysis bullosa hereditaria.

2. The onset occurs in infancy or early childhood.

3. The condition is strongly hereditary and therefore will be found in other members of the family. It is rarely acquired.

The cause is probably due to a congenital malformation in the elastic tissues of the upper derma. Normally the prolongation of the terminal fibers of this tissue between the cells of the basal layer probably constitute an important factor in binding the epidermis to the corium. In epidermolysis bullosa, there is an absence of these fibers in the papillary layer of the uninjured as well as the injured skin.

The patient who was shown demonstrates all these points. The onset occurred at 9 months of age when she began to walk. A younger sister had the first lesion at 13 months of age or three months before she began to walk. The members of the family who are affected develop bullae on their feet or

other points of pressure as soon as warm weather begins and suffer considerably till cold weather. The hereditary factor goes back as far as the grandfather can remember. Figure 4 is an incomplete chart showing the individuals affected.

NEVUS PIGMENTOSUS PILOSUS. DR. C. L. RUGGLES.

The nevus was shown because of its unusual size and the combination of three forms of the nevus—pigmented, hairy and fatty. All three forms were in evidence at birth. Three sources are considered the origin of the nevus cells:

Fig. 5.—Appearance of infant with nevus pigmentosus pilosus.

(1) the endothelium of the blood vessels and lymphatics; (2) mesoblasts and (3) epithelium.

Those who consider the epithelium the source believe that the pigmented malignant neoplasms occasionally originating in these cells are carcinomas and not sarcomas. It is thought that the embryonic impulse carries these cellular elements beyond the normal line of development. Most nevi remain stationary in size but occasionally they become larger, darker and more hairy. Electrolysis, caustics, carbon dioxide snow, radium and the roentgen rays are the dermatologic weapons. It is hoped that the portion of this large nevus which remains conspicuous when the hair is cut suitably and clothing worn can be rendered less of a blemish.

A Cod Liver Oil Requirement Curve. Dr. N. C. Wetzel.

From data already reported by Gerstenberger and Nourse, it has been found possible approximately to predict the minimal amount of cod liver oil required to prevent the development of rickets in the human infant. After it was concluded from the evidence at hand that the active development of rickets is directly related to a rapid gain in weight and the progress of healing to the unusual negative acceleration of growth, it was easy to choose suitable points for the approximate location of a curve describing the variation in dosage of cod liver oil with changes in the rate of growth. This is shown in figure 6, in which the zone between the diagonals $Q$ and $Q'$ represents the minimal pro-

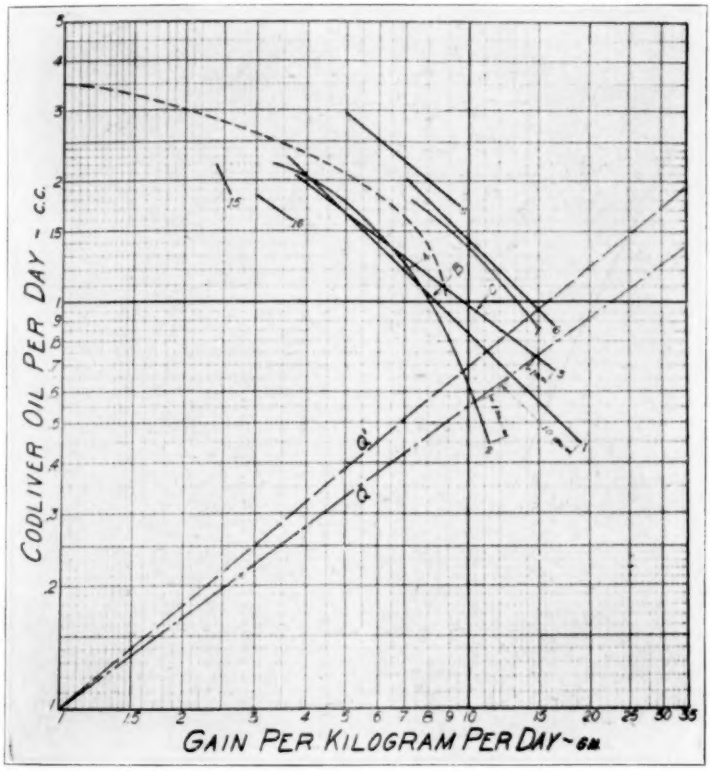



Fig. 6.—Curve showing the variation in dosage of cod liver oil with changes in the rate of growth.

tective dose of cod liver oil for diminishing unit rates of gain. The protective dose of cod liver oil that was used thus varies inversely with age, diminishing from 0.75 cc. of cod liver oil per day at birth to from 0.2 to 0.3 cc. at from 6 to 12 months of age. Recent observations have confirmed these estimates. This is a preliminary report.

Data Concerning the Dose of the Antirachitic Factor in the Prevention and Cure of Rickets. Dr. H. J. Gerstenberger.

In the study made by Gerstenberger and Nourse on the prevention of rickets in premature infants, it was learned that the amount of the antirachitic factor required to prevent and cure rickets was much smaller than is ordinarily accepted.

In order to ascertain how correct this conclusion was, another group of premature infants was fed small amounts of cod liver oil according to a tentative cod liver oil requirement curve and according to the weekly gain in weight per kilogram. Roentgenographic studies and chemical studies of the blood serum were made at frequent intervals to determine the presence or absence of pathologic changes.

Some infants were studied. All remained free from rickets except one who developed a mild form which disappeared on the same regimen as the child grew older, and at a less rapid rate. The average maximum daily amount given was 0.75 cc. and the minimum, 0.4 cc. Figure 7 is a graphic chart illustrating one of the cases.

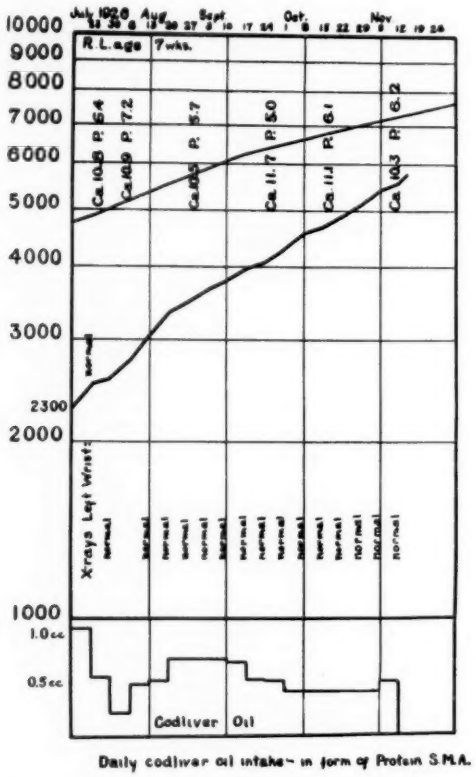



Fig. 7.—Effect of cod liver oil on infant with rickets.

In the cure of patients with rickets, observations made showed that the amount of cod liver oil given according to the curve used for the prevention of rickets and according to the per kilogram gain in weight was not sufficient to bring healing to completion with the same speed as was observed when the rachitic infant is exposed to the quartz lamp or is given S. M. A. Even a dose of 1 cc. of cod liver oil a day, which for some infants was much above the amount required, according to the plan followed, was adequate to bring healing to a satisfactory end only after a period of 240 days, in an infant with an active nonhealing form of rickets.

The amount of exposure to the actinic rays necessary to bring about healing to a decided degree was found to be surprisingly small. In one case an

exposure to the quartz mercury lamp at a distance of 80 cm. for a period of ten minutes was sufficient to start healing in a white infant after an interval of days and to continue the healing process, as recorded by the roentgenogram, for a period of days. When healing ceased in this infant, the rickets had healed approximately 50 per cent. This observation indicated the probability that the amount of actinic rays at present employed for the cure of rickets is much greater than is necessary. Observations regarding this point are in the process of being made.

RACHITIC SPASMOPHILIA. DR. J. I. HARTMAN.

The conclusion has been arrived at in this clinic that spasmophilia, as it ordinarily occurs, develops in rachitic infants who have undergone a peculiar type of antirachitic treatment. For this reason, I believe that the term "rachitic spasmophilia" should be used for these cases.

I believe that rachitic spasmophilia results from an interruption in the treatment of persons with rickets. It is well known that the incidence curve of spasmophilia shows two peaks, a greater one in the spring and a lesser one in the fall. It is my opinion that these peaks are brought about by the spontaneous interruption of the exposure of rachitic infants to sunlight. In the spring, when richets occurs most frequently and is most severe, the development of spasmophilia is due to the appearance of sufficient sunshine to influence the rachitic process, this increase occurring in an interrupted fashion owing to the changeability of the weather at this time of the year.

In the fall, because of the decrease in the amount of sunlight and the beginning of inclement weather, rachitic infants also are exposed in an interrupted manner to sunshine. These infants, as one would expect, are principally young ones, who, because of their age, have been kept away from the summer sun, and in the fall have been exposed irregularly to sunlight.

I have seen the development of spasmophilic symptoms in one case of rickets after a single exposure to the quartz ultraviolet light in which the healing was sufficient to show in the roentgenogram.

HEART BLOCK IN DIPHTHERIA WITH RECOVERY. DR. ELIZABETH BARTOS.

A white girl, aged 8 years, was brought to the hospital on the third day of her illness with nasal and pharyngeal diphtheria.

On admission the child was deeply toxic, with extensive membrane covering the tonsils and the postpharyngeal wall, and the interior of both nostrils. The cervical glands were markedly enlarged. The heart seemed normal except for a soft systolic murmur at the apex. The pulse rate was 120; it had a fair volume and was regular. The temperature was 38.4 C. (101.1 F.).

She had received 10,000 units of antitoxin a few hours before admission, and 30,000 more units were given after admission.

The patient was kept under the influence of morphine, and a 5 per cent solution of dextrose was administered intravenously and per rectum several times daily. She improved gradually. The membrane became absorbed by the ninth day; the pulse was regular, the rate around 100, and an electrocardiogram taken on this day showed a normal mechanism with slight tachycardia. The P-R interval was 0.14 second.

On the morning of the thirteenth day of illness, the patient vomited once, became cyanotic and was in apparent distress; the pulse was found to be irregular, around 100 or 120, and then suddenly it dropped to about half of this rate.

An electrocardiogram taken immediately, showed a two to one block, with an auricular rate of 140 and a ventricular rate of 70; the P-R interval was 0.2 second.

Absolute rest and morphine were ordered again. The block lasted about six hours; then the pulse rate became from 104 to 110, with numerous extra-

systoles. These were present though decreasing in number until the eighteenth day of the disease. From then on the pulse remained regular, and was of good volume, and the electrocardiogram was normal. The patient was discharged on the twenty-eighth day in a good general condition with a regular pulse, a normal heart and a normal electrocardiogram. When seen again six months later, she seemed to be in perfect health, and cardiac pathologic change was not evident.

ALLERGIC MANIFESTATIONS IN TUBERCULOSIS. DR. C. W. BURHANS.

When a person is infected with tubercle bacilli his tissues soon become sensitized to the protein of this organism, and the altered reaction of the tissues was called by Von Pirquet, "allergy." By the time tuberculosis is diagnosed clinically, it is in all its forms influenced by this allergy but the manifestations of allergy vary a great deal. Allergy is a defense reaction which attempts to prevent the dissemination of tubercle bacilli by surrounding tuberculous foci with an exudate. Marked allergic reactions, however, may of themselves produce severe toxic symptoms and even death.

In children, manifestations that have been considered extensions of the tuberculous process are often only allergic exudates. An attempt should be made from a clinical standpoint to interpret them in this light, because they may have an important bearing on the prognosis. The belief is expressed that the difference between so-called "splenopneumonia" or "epituberculosis" and caseous pneumonia is purely relative.

Slides were shown of the roentgenograms of the chest of a tuberculous infant who suddenly developed a dense infiltration of the upper lobe of the right lung. The child gained in weight at a more rapid rate after the infiltration appeared than before. The infiltration disappeared after a few months.

CONGENITAL ANEMIA. DR. J. E. MCCLELLAND.

The case of an infant, aged 4 months, who to all outward appearances was in perfect health was reported. The case was presented, not because it was one of the rare and interesting forms that so often are encountered in the medical literature, but because Dr. McClelland had a suspicion that congenital anemia is more common than the paucity of reports would lead one to believe.

Five cases have been reported in the literature, two in Germany and three in this case.

The case described conforms closely to the others. The parents have been healthy and usually young.

One case occurred in a secundipara, and all the others in primiparas. According to the histories, none of the mothers had had anemia, tuberculosis or syphilis, nor had they taken drugs or had toxemia or any severe illness. The striking thing was that the infant was born at full term, of normal weight, and was pale.

The only important factor discovered on examination was that four of these five infants had a large spleen which could be readily palpated. In one case a large liver was reported, but not in the others.

The blood picture was characteristic. It was similar to that encountered in the primary anemias of the adult. The red cell counts were usually between 1 and 2 million. The hemoglobin was higher in proportion, usually about 50 per cent, and gave a high color index, about 1.3. The differential counts were not instructive as far as the proportion of the different elements were concerned, but they were helpful in that they showed some myelocytes and 2 or 3 per cent of neutrophilic myelocytes, the nucleated red cells numbering about 10 in a white cell count of 200. Slight anisocytosis also was present.

Cases in which the bleeding time or the coagulation time has been prolonged, or in which hemorrhagic disease of the new-born occurred, were not seen. The prognosis apparently is good.

One infant died on the tenth day. All the rest recovered. Two of the infants received intravenous transfusions and recovered. Recovery is usually complete at the end of three or four months, at the most at six months.

FOREIGN BODY IN THE BRONCHUS OF A CHILD WITH CONGENITAL ABSENCE OF THE OTHER LUNG. DR. O. L. GOEHLE.

Total absence of one lung is rare. About twenty cases have been reported, only twelve of them, however, in detail. All cases were diagnosed at autopsy only.

In a child who was first seen at the age of 8 months, a diagnosis of dextrocardia and probable fibrosis obliterans of the right lung was made at the

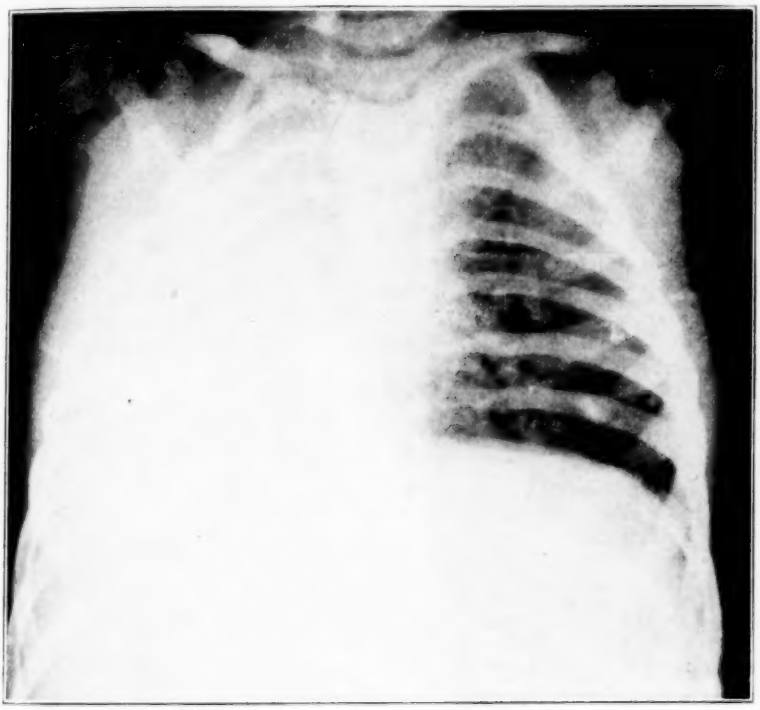


Fig. 8.—Roentgenographic appearance of chest of child with foreign body in the bronchus and congenital absence of the other lung.

hospital from physical examination as well as flouroscopic and roentgen-ray observations. Progress in both physical and mental development was satisfactory during the period from 8 months to 2 years of age, when the child was again brought to the hospital, this time in emergency.

While chewing gum and eating peanuts at the same time, the child had insufflated both and had begun to choke. Urgent symptoms of respiratory obstruction supervened. Breath sounds over the right side of the chest were absent; over the left side of the chest they were heard only occasionally. Flouroscopic and roentgen-ray examination of the chest did not show the presence of a foreign body. The child died within twenty minutes after the onset of the attack.

An enormous lung filled the entire left side and part of the right side of the chest. The heart occupied the lower two thirds of the right side of the

chest. An enormously enlarged thymus filled the upper right side. There was total absence of the right lung. The left bronchus was almost completely blocked by large piece of gum and two large pieces of peanut kernel. The abdominal organs were in their normal position.

INSPECTION OF THE NEW BABIES' AND CHILDREN'S HOSPITAL.

A tour of inspection of the Babies' and Children's Hospital was made with the assistance of the visiting and house staffs of the hospital. Some of the points especially emphasized were the following:

1. The living rooms, research laboratories and library on one floor.
2. The sound proofing of wards, corridors, diet kitchens and utility rooms.
3. The combined use of terrazzo and rubber flooring in corridors and wards.
4. The humidifying apparatus automatically operated in the wards.
5. The location of the contagious division on the second floor of the six story building with a separate elevator and a separate admission entrance.
6. A liberal equipment for heliotherapy and fresh air therapy by the use of porches on the south and north sides and the roof.

*Friday Afternoon Session, Auditorium, Cleveland Medical Library, Oct. 14, 1927.*

Juvenile Acrodynia. Dr. C. W. Wyckoff.

Cirrhosis of the Liver in Infancy: Report of Two Cases in One Family. Dr. Harold O. Ruh and Dr. Paul G. Albrecht.

Cleidocranio Dysostosis. Dr. Warren C. Fargo.

Chronic Diarrhea of Unusual Interest. Dr. E. R. Brooks.

Froin's Syndrome Due to Syphilis in a Boy Aged Thirteen Years. Dr. C. D. Dauer.

Exophthalmic Goiter. Dr. A. F. Furrer.

The Use of Concentrated Breast Milk for Premature Infants. Dr. J. R. Thompson.

Psychiatric Approach to Enuresis. Dr. O. B. Markey.

Infantile Eczema. Dr. D. C. Mebane, Toledo, Ohio.

A Case of Hirschsprung's Disease Four Years After Operation. Dr. R. R. Rogers, Warren, Ohio.

A Résumé of the Mansfield and Richmond County Child Health Demonstration. Dr. Charles L. Shafer, Mansfield, Ohio.

JUVENILE ACRODYNIA. DR. C. W. WYCKOFF.

Juvenile acrodynia is an old disease rediscovered, but there are so many salient points of difference between the acrodynia in adults that was first described in the early part of the nineteenth century and these cases found in the past decade that the author believes that the term "juvenile acrodynia" should be used for the type found in children.

*Etiology.*—Only by the process of elimination of other factors can one say that a subacute infection is the most likely progenitor of the more or less chronic toxemia found in these cases. Nearly all writers on this subject mention infection of the upper respiratory tract as a precursor and as an accompaniment of this disease.

*Symptoms.*—The cardinal symptom is a peculiar dull redness and swelling of the hands and feet with a moderate degree of desquamation and a cold, clammy feeling. Various rashes appear on the skin, not unlike those of the different acute exanthemas. Marked irritability and restlessness, sleeplessness and other symptoms indicating a general peripheral polyneuritis affecting the sensory and trophic nerves occur. Anorexia is marked, sometimes complete. Asthenia is a constant symptom probably due to a great degree of muscular hypotonicity. A varying degree of photophobia is present.

The ages at which the children are affected are in most instances from 12 months to 4 years. The duration of the disease is from three to eighteen

months. The mortality is low. The few deaths reported are due to some intercurrent infection and the low physical ebb of the body, because of insufficient food.

*Treatment.*—There is nothing to do except to keep the child nourished, if possible, and on a rational sick diet for its particular age. It should be made to eat or, if necessary, gavage should be resorted to and continued sometimes for weeks. Tonsillectomy and adenoidectomy have been recommended, if the disease is not too far advanced. Drugs of any description are useless.

CIRRHOSIS OF THE LIVER IN INFANCY: REPORT OF TWO CASES IN ONE FAMILY. DR. HAROLD O. RUH and DR. PAUL G. ALBRECHT.

Two cases of infants in one family with cirrhosis of the liver were reported. The etiology was not determined, the parents having been especially well, without a history of tuberculosis, syphilis or alcoholism. During both pregnancies, the mother had not shown abnormal symptoms or illnesses.

Both infants were born at full term, with normal deliveries; they were breast fed and during the first three months of life were apparently normal.

M. A. was first seen at the age of 6 months on account of loss in weight and irritability. The liver and spleen were found to be enlarged. This enlargement progressed; the skin became icteric, ascites developed, emaciation became pronounced, and death occurred at the age of 9 months. The autopsy revealed cirrhosis of the liver (Laennec's type).

H. A. was first seen at the age of 2 weeks. The physical examination did not show any abnormalities. The liver and spleen were normal. The infant progressed normally until it was 2 months of age, at which time vomiting occurred. The stools and urine were normal.

The Wassermann and tuberculin tests were negative, and the blood was normal. When the child was 3 months old, the liver was 3 cm. below the costal margin in the midclavicular line, and the spleen was palpable. A month later, the liver was 5 cm. below the costal margin and the spleen considerably enlarged. At the age of 5 months, the liver was 9 cm. below the costal margin.

From this time until death, there was a progressive loss in strength. Ascites and icterus developed similarly to that in the other child. Death occurred at 9½ months. Autopsy confirmed the diagnosis of cirrhosis of the liver (Laennec's type). The rare occurrence of this condition in infancy and especially of two cases in one family without known etiology is of unusual interest.

CLEIDOCRANIO DYSOSTOSIS. DR. WARREN C. FARGO.

Seventeen cases are reported in three generations, in which the oldest generation is of English descent. Patients with typical forms of the disease are of small stature, have sloping shoulders and globular-shaped heads. Many are mentally retarded.

The fontanels and sutures are wide at birth and close slowly, the fontanels often being open in adult life, when deep sulci following the suture lines separate prominent bosses. Dentition is slow; the teeth are irrgular, and the palate is usually highly arched or cleft.

The clavicles vary widely, from marked angulations or small areas of fibrous union in them to complete absence of one or both, the usual deformity being a sternal fragment with or without fibrous attachment to the acromion process.

Depression of the sternum, coxa valga, genu valga, compression of the pelvis and shortening of the second phalanges of the toes are described in the literature. Attention is called to incomplete calcification, as demonstrated by the roentgenogram of several cervical, dorsal, and lumbar neural arches, of the symphysis pubis and the body of the pubis or the ischium. On palpation, these bones are firm and feel normal.

The condition is hereditary and familial; it is found in both sexes and may be transferred by either parent. The picture seems to be that of a general

constitutional defect of development of the bones of unknown cause, with predominance in certain groups of bones, many of which represent midline embryologic variations.

CHRONIC DIARRHEA OF UNUSUAL INTEREST. DR. E. R. BROOKS.

In D. S., a girl, aged 12 years, who had been suffering from chronic ileocolitis of unknown origin for the last two and a half years, the illness began with fever, abdominal pain and from fifteen to twenty loose, watery stools daily which later contained considerable gross blood.

Roentgen-ray examination of the chest was negative for tuberculosis; examination of the stool did not show tubercle bacilli, but many bacteria and pus; a smear showed a predominating gram-positive bacillus. Parasites were not seen. An intradermal test was negative in a 1:10 dilution to both bovine and human tuberculins.

On Jan. 12, 1927, examination of the blood showed: red blood cells, 1,475,000; white blood cells, 4,200; hemoglobin, 30 per cent; color index, 0.9; the differential count showed: polymorphonuclears, 51 per cent; small mononuclears, 41 per cent; large mononuclears, 4 per cent; one nucleated red cell, and marked poikilocytosis.

After two transfusions of 500 cc. of whole blood, the blood count on March 9 showed 5,370,000 red cells and hemoglobin, 65 per cent. Proctoscopic examination did not show ulcers; the intestinal mucosa was pale and edematous throughout and was not atrophic but one hemorrhagic spot was present rather high up. The weight ranged from 36½ pound (16.5 Kg.) in March to 51 pounds (23.1 Kg.) in October.

*Treatment.*—The diet consisted of milk curd and lactic acid milk, concentrated S. M. A., casein milk with malt soup extract, and, finally, two quarts of whole milk boiled down to one quart, 100 Gm. of raw meat balls three times a day, beef broth, ice cream, jello, zwieback, juice of 1 quart of strawberries daily and, later, eggs and a little vegetable were added.

Exposures to ultraviolet rays were given for a long period; several blood transfusions were given during the early part of the hospital course. Internally, bismuth, kaolin, iron and camphorated tincture of opium were employed at different times, and large amounts of yeast powder were also administered. Olive oil enemas and starch and hot water irrigations were also employed.

FROIN'S SYNDROME DUE TO SYPHILIS IN A BOY AGED THIRTEEN YEARS. DR. C. C. DAUER.

A boy, aged 13, was admitted to the hospital because of paralysis of his right arm and weakness of his legs. Six weeks previously, he had noticed some weakness of the right arm which became progressively worse. He finally was unable to use this arm except for flexion and supination. The muscles of his legs became weak, and he was able to walk only with difficulty. Loss of control of the rectum or bladder did not occur.

On examination, he showed a paralysis of all the muscles of his right arm except the biceps and the supinators. The muscles involved were those supplied by nerve fibers from the sixth, seventh and eighth cervical, and the first dorsal segments of the spinal cord. There was little volitional control of the muscles of the legs, but paralysis was not present.

Lumbar puncture showed an icteric fluid not under increased pressure. The fluid coagulated a few minutes after withdrawal. Combined lumbar and cisternal punctures (Ayer test) were performed, and manometers were connected to the needles. Pressure on the jugular veins caused a marked rise in pressure in the cisterna but showed only a slight rise in the lumbar region. This indicated a block somewhere between the two needles. One cubic centimeter of iodized oil 40 per cent was injected into the cisterna, and a roentgenogram was taken of the cervical and dorsal spine. This roentgenogram showed that the iodized oil had stopped at the level of the disk between the fourth and fifth cervical vertebrae.

With such a history and physical manifestation, one would suspect a tumor of the cervical cord located on the anterior surface. However, there was a family history of syphilis, the mother having tabes dorsalis and a sister, interstitial keratitis. The spinal fluid of the boy was anticomplementary, but the blood showed a four plus reaction. Antisyphilitic treatment consisting of neoarsphenamine, tryparsamide and potassium iodide was instituted, and in the course of two months the boy showed a complete recovery from the paralysis.

EXOPHTHALMIC GOITER. DR. A. F. FURRER.

A girl, aged 11 years, in whom the onset and development of symptoms had been insidious, was under medical supervision in the home for seven weeks and was then studied at the Babies' and Children's Hospital for another month. Thyroidectomy was performed at Lakeside Hospital.

The literature to date was reviewed.

THE USE OF CONCENTRATED BREAST MILK FOR PREMATURE INFANTS. DR. J. R. THOMPSON.

A report is made of the results of feeding concentrated breast milk to a group of thirty-seven premature infants. The concentrated breast milk is fed only to those infants who will not take a sufficient amount of breast milk or of whatever type of food is available and suitable to enable them to grow normally. It was found that one of the problems in caring for the premature infant is to get it to take a sufficient quantity of food.

The number of feedings have been limited to five in twenty-four hours, because it is believed that increasing the number of feedings defeats the purpose of getting sufficient calories into the infant. The amount given is never greater than the amount that they will take without urging. Some of these infants, however, receive as high as 400 calories per kilogram.

Seven of the thirty-seven infants died, and five of them were in the hospital less than one week. Most of the infants required an energy quotient of 250 before they would gain.

The concentration was carried as far as necessary, up to a maximum of 4:1, for instance: if an infant would not take more than 1 ounce (30 cc.) at a feeding and would not gain, he was given an ounce of 2:1 concentrated breast milk; if he still did not gain and would not take a greater quantity, the concentration was increased to a 3:1 ratio and in some instances to a 4:1 ratio.

The idea in using concentrated breast milk is to get sufficient calories into the infant and to do this by giving him individual food elements in the same proportion as they exist in breast milk.

QUESTION: Does the milk caramelize in concentrating and is there any harm in that?

DR. THOMPSON: It does sometimes. I discovered one batch of breast milk that caramelized markedly, and I could not understand it. I had the milk analyzed and discovered that there was 15 per cent fat in it; I was feeding the baby 30 per cent fat, and when there is high fat in the milk, it will caramelize.

QUESTION: Did it do any harm?

DR. THOMPSON: To this infant it did.

PSYCHIATRIC APPROACH TO ENURESIS. DR. O. B. MARKEY.

Enuresis is approached largely as a problem involving the emotions, the organic factor being brought up only when it more or less directly influences the personality. The discussion opens with a description of urinary elimination from birth on. Conscious control of elimination must be acquired in order for the person to make social and personal adjustments. In the infant, the pleasure of elimination occasionally becomes the motive for continuing it as

the child grows older, and becomes an unconscious reason for clinging to the uninhibited privileges of babyhood. In addition, the infant voids in a recumbent position. Delay in training him to void in an upright positon and by voluntary control often results in a fixed conditioning to infantile elimination. Occasionally a child regresses to an infantile level, and enuresis becomes a symptom.

The following causes for development of enuresis as an emotional problem are discussed:

(*a*) Delay in training.

(*b*) Bladder consciousness. Conscious control of an unconscious urge is weak and fleeting, yet certain of these children vainly try to control this symptom by their will. Some of them continue because they think their kidneys are weak and others because they have been told that they will continue for some time, until puberty, for instance.

(*c*) Stigmatizing a child in treatment of this trouble.

(*d*) Emotional discipline, in general or in regard to the enuresis.

(*e*) A child's conscious drive for attention.

(*f*) Psychosexual disorders, in which sexual imagery or masturbation, etc., play a part. Ejaculation is said to be an associated form of elimination.

(*g*) Bladder tics.

(*h*) Emotional conflicts resulting from a child's struggle against this problem.

The principles of treatment include the following:

(*a*) First of all, the elimination of the organic factor as a general or local cause.

(*b*) The consideration of each child as an individual suffering from an emotional disorder which must be approached through a study of the child in all of its phases.

(*c*) The avoidance of repressing methods, even though they succeed in some instances.

(*d*) Suggestion works in some cases, as by placebo medication, positive direct suggestion, or the chart method.

(*e*) Selected cases can be approached individually by a mutual review of the causes and their gradual elimination, an opportunity for the child to talk the thing over with a doctor in whom he has faith and whom he does not fear.

(*f*) Change of environment.

(*g*) Elimination of associations suggesting enuresis, such as cold rubber pads, segregation, etc.

(*h*) An objective psychiatric approach is necessary and possible for any physician and is essential in every case. When such an approach is used, it will be seen that persistent cases of enuresis are in almost every instance associated with serious disorders of behavior and personality which have also to be approached.

(*i*) The whole question of habit training must be more or less clearly associated in the physician's mind.

INFANTILE ECZEMA. DR. D. C. MEBANE.

The author cites instances of infantile eczema to show how insufficient fluid intake, mild infections, such as colds, nasopharyngitis, otitis media or pyelitis, and overfeeding may initiate eczema or cause cured eczema to reappear.

He states that the well balanced diet that is sufficient in the food essentials, high enough in calories to bring about an average gain in weight, and with added food accessory factors not to permit of deficiency is the fundamental principle in preventing and curing infantile eczema. He thinks that if sufficient fluid intake, the avoidance of infections and good general hygienic care including sunlight, fresh air and proper care of the skin are added to these measures, infantile eczema would be much less frequent and rarely severe.

He decries the routine feeding of concentrated acidified milk. Infants so fed are forced to digest and metabolize much more food than the body needs and frequently develop eczema.

The author thinks it is a mistake to take one food principle out of the diet. In feeding experiments he finds carbohydrate, fat or protein interchangeable. Frequently, the supposedly offending principle can be increased, and the other principles lowered with a resulting disappearance or improvement in the eczema.

A Case of Hirschsprung's Disease Four Years After Operation. Dr. R. R. Rogers.

A case originally reported (*Ohio State M. J.* **19**:172 [March] 1923) as secondary megacolon is reviewed. For the first year of life the infant in question suffered from marked anal atresia, which was remedied at 1 year of age by a plastic operation on the anus. A short time later, it was discovered on exploratory operation for obstipation that the infant had megacolon. The point of interest was whether this was congenital, along with the anal atresia, or whether the dilatation and hypertrophy of the colon was secondary. This paper is a report on the same case six years later when the patient was 7 years old. He still had all the symptoms and roentgenographic manifestations of megacolon in spite of having had a fairly normal anal sphincter for six years. The conclusion is that the megacolon was congenital rather than acquired.

A Résumé of the Mansfield and Richmond County Child Health Demonstration. Dr. Charles L. Shafer.

A brief outline was given of the purpose, methods and accomplishments of the Child Health Demonstration in Mansfield and Richmond County, Ohio, in the period from January, 1922, to January, 1926. The central idea was to assist a typical industrial and agricultural county to develop protective and educational measures for the health of its children as broadly and soundly as possible.

Since the beginning of the demonstration, the public health administration has been put on a sound full time basis; public appropriations for health work have more than trebled in amount; the adoption by the Board of Education of supervision of health education in the schools has been accelerated; ten infant health centers have been established, which give every promise of continuing; there has been a wide dissemination and correlation of health knowledge among parents and children; there has been an increased demand for preventive service from the physicians, who have responded well to the demand; there was an increase of children accepting physical examination in the schools from 8.2 per cent in 1921-1922 to 85.5 per cent in 1925-1926. Much of the work in the schools was aided by the Blue Ribbon Project or First Premium for Health, of which 3,630 were awarded in 1926-1927.

*Saturday Morning Session, Auditorium, Cleveland Medical Library, Oct. 15, 1927.*

Hydrocephalus. Dr. C. E. Locke, Cleveland Clinic.

Anomalies of the New-Born. Dr. C. A. Hamann, Professor of Applied Anatomy and Clinical Surgery, Western Reserve University.

Harelip and Cleft Palate, Dr. S. W. Kelley.

The Physical Growth of Children. Dr. T. Wingate Todd, Henry Willson Payne Professor of Anatomy, Western Reserve University.

Familial Partial Defect of Cranial Bones. Dr. A. C. J. Brickel, Clinical Instructor in Anatomy, Western Reserve University.

Bicameral Dextrocardia with Systemic Pulmonary Circulation. Dr. Justin A. Garvin.

Diaphragmatic Paralysis Resulting from Injury of the Brachial Plexus. Dr. J. W. Epstein.

Lymphatic Tumor of the Intestine Causing Obstructions. Dr. Hugh J. Leslie.

Sarcoma of the Ileum with Partial Obstruction. Dr. F. W. Linn.

Studies in Smallpox. Dr. R. B. Hauver.
Diphtheria Carriers. Dr. R. M. Fullerton.
Periostitis of the Fingers and Toes of Unknown Etiology. Dr. A. G. Evans.
Self-Selected Diets of Newly Weaned Infants. Dr. Clara Davis.

HYDROCEPHALUS. DR. C. E. LOCKE.

That hydrocephalus has existed since early times has been evidenced by the discovery of hydrocephalic skulls in Egyptian cemeteries. Among early authors who described this condition are Hippocrates, Heironymus and Vesalius.

The function of the cerebrospinal fluid must be kept in mind in order to understand the causes of hydrocephalus. The formation, circulation and absorption of the cerebrospinal fluid were discussed and illustrated by slides showing celloidin casts of the cerebral ventricles and of the cerebral subarachnoid spaces. The cerebrospinal fluid pathways may be divided into the intraventricular and the subarachnoid systems. Obstruction of either of these systems produces internal hydrocephalus.

Various types of hydrocephalus were discussed and cases presented, with accompanying lantern slides.

Internal hydrocephalus is usually associated with infratentorial tumors and frequently with suprasellar growths. Here the obstruction is in the ventricular system and may be relieved by removal of the tumor, or, in the former case, simply by suboccipital decompression. In hydrocephalus occurring with chronic meningitis or following an intracranial hemorrhage at birth, the obstruction is in the subarachnoid system. Illustrative cases of this type were shown in which relief was obtained by a surgical opening made between the internal subarachnoid channel and the lateral ventricle. Two cases of internal hydrocephalus produced by arachnoiditis of the posterior fossa were shown in which the patients were cured by a surgical reestablishment of the foramen of Magendie. Patients with congenital hydrocephalus and hydrocephalus associated with spina bifida were also described.

From a surgical standpoint there is much to be learned about the treatment of patients with hydrocephalus; however, certain forms of it may now be relieved by operation.

ANOMALIES OF THE NEW-BORN. DR. C. A. HAMANN.

Lantern slides were shown depicting many common and several rare conditions found in the new-born, and a discussion of them was presented. Pictures of the cases or specimens are given in table 2.

*The Skin*

Milroy's disease
Sclerema neonatorum
Fetal dropsy
Congenital elephantiasis
Dermoids

*The Neck*

Branchial Fistulas and Cysts
Cysts of the Thyroglossal Ducts
Congenital Cystic Hygroma

*Genito-Urinary System*

Supernumerary penis
Hypospadias
Cryptorchism
Ectopic kidney
Congenital cystic kidney
Renal tumor
Exstrophy of bladder

*Nervous System*

Encephalocele and Meningocele
Spina bifida

*Gastro-Intestinal Tract*

Congenital pyloric stenosis
Intussusception
Congenital ascites
Hirschsprung's disease
Imperforate anus

*Bones and Joints*

Congenital absence of various bones
Polydactylism
Syndactylism
Radio-ulnar synostosis
Clubhand and foot
Congenital elevation of scapula
Osteogenesis imperfecta
Congenital tumors of the sacrococcygeal region

HARELIP AND CLEFT PALATE. DR. S. W. KELLEY.

Only five points were emphasized:

1. Do not operate until the patient is in a suitable condition and the dangers following closely on birth are past, regardless of the urging of chagrined parents or of impatient surgeons.

2. In the meanwhile: (*a*) secure nutrition and healthy tissues, and (*b*) narrow the cleft.

3. Nature tries to finish her uncompleted handiwork. One should work with her. Employ the muscles to help the closure; also protect the membranes.

4. If, instead of having been hurried into operation, the patient has been neglected, and not operated on during infancy, "it is never too late to mend" the lip or the palate.

5. After operation on the palate, training in speech is indispensable. Good speech can be secured if the muscles of the palate are preserved.

Lantern slides show the method of strapping across the complete cleft of the lip and palate, resulting in narrowing of the cleft preparatory to operation. Strapping also promptly hides the deformity, stops the inrush of cold air and dust which produce rhinitis, and enables the infant to nurse better. An obturator nipple facilitates sucking.

Clefts of the lip and palate in young children, in older children and in adolescents were shown before and after successful closure.

THE PHYSICAL GROWTH OF CHILDREN. DR. T. WINGATE TODD.

So long as one continues to estimate growth quantitatively, in terms of dimensions like height and weight, the problem of growth must remain complex and obscure, for dimensions are readily modifiable by many factors. The qualitative method, however, promises a much more reliable criterion, but it is more difficult to learn.

The method used by the author is the observation of the epiphyseal ends of the bones: it is a roentgenologic study and results are based on the records of 800 children, most of them examined at least twice over a period of eighteen months, of 1,500 human skeletons of known age and 700 skeletons of growing mammals.

There is sufficient evidence to demonstrate that the mere appearance of a center of ossification in the epiphysis is not accurately timelinked. In contrast with this, the later stages of etching out the final bony surfaces of the epiphysis are definitely timelinked, and it is on these that we depend for our criteria.

The knee, hand and elbow have been analyzed in detail, and the results are demonstrable. Details of growth of the knee will be found shortly in *Northwest Medicine*.

FAMILIAL PARTIAL DEFECT OF CRANIAL BONES. DR. A. C. J. BRICKEL.

A colored boy, aged 8 years, had elliptic openings, measuring 7 by 5 cm., in the posterior part of the parietal bones. They were symmetrical and equal, and were present in the grandfather and father also. The grandmother settled a dispute concerning the child's claim of parentage by seeking this peculiarity in the child's skull.

There were no symptoms except some mental retardation. The scalp pulsated over the openings and was sensitive.

BICAMERAL DEXTROCARDIA WITH SYSTEMIC PULMONARY CIRCULATION. DR. JUSTIN A. GARVIN.

A girl, apparently normal at birth, nursed less well at 2 weeks of age and had choking spells. When she was 1 month old, a physical examination showed a right sided heart. The roentgen ray confirmed this and showed the stomach on the left side. Precordial activity was visible to the right of the sternum, and the heart beat was palpable as far as the right midaxillary line. A loud

blowing murmur, loudest at the base and fading toward the apex, was audible over the entire chest.

Death occurred from pneumonia at 2 months of age.

Autopsy showed a right sided heart consisting of one auricle and one ventricle. The aorta arose from the ventricle in the normal way, but the pulmonary aorta was absent. The circulation through the lungs was found to be systemic, like that of the kidneys. Two pulmonary arteries arising from the descending thoracic aorta carried the blood to the lungs, from which it was returned by the pulmonary veins which emptied through a curved extension of the superior vena cava into the auricle. The liver was greatly enlarged, and the spleen was absent.

Diaphragmatic Paralysis Resulting from Injury of the Brachial Plexus. Dr. J. W. Epstein.

This paper was published in full in the American Journal of Diseases of Children (**34**:634 [Oct.] 1927).

Lymphatic Tumor of the Intestine Causing Obstructions. Dr. Hugh J. Leslie.

This patient was first examined at the age of 3 weeks, when a complaint of failure to gain and constipation was made. The infant gained readily on simple formulas, but was constipated at the age of 6 weeks and had an attack of projectile vomiting, which lasted more than twelve hours. The vomitus consisted mainly of bile with slight fecal odor. Several enemas were given during this time without results. The rectal temperature was 98 F. A mass was palpable in the right iliac region. A diagnosis of intussusception was made and the child sent to the hospital. While the abdomen was being examined under anesthesia, the patient passed a large yellow stool, which prevented a diagnosis. Rectal examination showed a freely movable, lobulated, sausage shaped mass.

At operation a coarsely lobulated, tumor-like mass, about 8 by 5 cm., was found adhering to the ileum.

A resection was done, with an uneventful recovery.

Sarcoma of the Ileum with Partial Obstruction. Dr. F. W. Linn.

A boy, aged 8 years, has been complaining of cramplike pains in the abdomen for the past two months. These attacks occurred as often as fifteen times daily; they would cause him to get up and walk about, and were sometimes relieved if he would lie across the father's knees. The ingestion of food often seemed to incite the attacks. Vomiting, blood in the stools or constipation did not occur. The patient had lost 15 pounds (6.8 Kg.) during the two months.

Early in the disease, abdominal tenderness was not present nor was any mass palpable. The remainder of the physical examination revealed nothing abnormal. Later, there was tenderness in the region of the spleen and still later around the umbilicus. Free fluid could not be made out.

A barium meal passed through the intestine freely. However, while a barium enema was being given, a peculiar formation like a capital M could be seen with the fluoroscope at about the junction of the ileum and the ascending colon. A diagnosis of intussusception was made and the patient immediately operated on.

An irreducible intussusception was found, and a part of the ileum, the cecum and the appendix were resected. The mesenteric glands were enlarged.

A tumor mass, 1¼ inches (3.1 cm.) in diameter, was found in the wall of the ileum. Some ulceration of the mucosa was present, and the deeper layers of the ileum were involved by a large sheet of lymphoid cells. The tumor was vascular. A microscopic diagnosis of lymphosarcoma was made.

The patient had an uneventful operative recovery but death occurred two months later, apparently from metastasis.

STUDIES IN SMALLPOX. DR. R. B. HAUVER.

This paper contains the conclusions of work done by Toomey, Gammel, Shafer, Dolch and Hauver on convalescent smallpox serum.

I was able to confirm the presence of virulicidal antibodies in convalescent smallpox serum, which were fairly stable in character. Four hours of incubation or four hours of inactivation do not destroy this antibody factor, nor does combined inactivation and incubation for the same length of time. This serum antibody appears from nine to twenty days after the symptoms of the disease begin and from seven to fourteen days after the rash starts. This virulicidity is not a property of the dermis, as washed sections of skin from patients convalescing from smallpox lack this virulicidal factor.

Convalescent smallpox serum was used therapeutically without any marked results.

It was attempted actively to immunize a calf by repeated injections of smallpox virus intradermally as well as subcutaneously; the serum obtained from this animal three months after the injections began was potent enough to prevent vaccinations in as low a dilution as 1:25 and seemingly to modify the course of the smallpox lesions in the few cases in which it was used, suggesting a therapeutic possibility in serums thus obtained.

DIPHTHERIA CARRIERS. DR. R. M. FULLERTON.

For five years, diphtheria carriers admitted to the contagious division of the Cleveland City Hospital have been subjected to various types of treatments and records kept of the results. The treatments used have been alkaline sprays, local applications of aniline dyes, gentian violet, acriflavine and mercurochrome-220 soluble, tincture of iodine, iron chloride, potassium chlorate, 5 per cent, menthol sprays, silver nitrate, an organic iodine preparation (iodoxybenzenepyridine sulphonate) in various percentages as a powder spray or in solution, tonsillectomy, adenoidectomy, plain water irrigations and no treatment at all.

The conclusions are that any local application or irrigation, even though it may be merely plain water, tended to render the carrier free from the bacillus a little sooner (from two to eight days) than control carriers who were left alone. In the majority of cases in which treatment was instituted, the condition cleared up from fourteen to thirty-one days after admission. Patients become bacillus-free sooner after tonsillectomy and adenoidectomy than with any other type of treatment, and for persistent forms, this is the best method to follow. When tonsillectomy and adenoidectomy fail, treatment does not do much good, the condition in the controls clearing up when nothing is done as quickly as when elaborate treatment is employed. Many of these patients have latent infections of the sinuses, and these respond to proper treatment of the nose and throat.

PERIOSTITIS OF THE FINGERS AND TOES OF UNKNOWN ETIOLOGY. DR. A. G. EVANS.

A white girl, aged 10 years, came to the Lakeside Dispensary complaining of swelling of several fingers and toes.

The father suffered from some chronic malady of one leg, probably a chronic osteomyelitis.

During the preceding winter, the patient had several attacks of sore throat. In March she had an attack of torticollis, with swelling of the cervical glands and fever. This was followed in a weak or ten days by swelling of the first and third fingers of the right hand and the third finger of the left hand and the fourth toe of the right foot and the right sternoclavicular joint. Moderate pain was present, especially at night; motion was little affected. The condition soon began to subside spontaneously but slowly. The tonsils were removed in April.

Physical examination revealed nothing of importance except for the swellings noted above and the scar behind the right ear. The tibias appeared normal. The involved digits were moderately enlarged and spindle shaped, with free motion, and they were not tender. The skin of the parts was not discolored.

Both the Wassermann and the intracutaneous tuberculin tests (1:1,000) were negative.

The roentgenogram shows a moderate lineal periosteal thickening of the first and third proximal phalanges of the right hand, third proximal phalanx, left hand; and fourth proximal phalanx, right foot. The joints are clear and there is no bony change. No further roentgen-ray or laboratory work has been possible.

The process has now receded to the point where the digits are nearly normal. Therapy was not instituted early.

SELF-SELECTED DIETS OF NEWLY WEANED INFANTS. DR. CLARA DAVIS.

A report illustrated with moving pictures was made of an experiment with three infants. At the time of weaning, breast-fed infants were taken from the breast suddenly and before them were placed trays containing natural, unselected foods, without mixes, without seasonings or condiments, or any alteration except cooking in the simplest manner; these infants were allowed to select their own foods without direction, in the quantity they wished, without suggestion from any one and without any outside influence. This was done to ascertain whether infants under these conditions would choose sufficient quantities of the various foods to maintain themselves and grow normally, and especially whether the choice would be vegetarian, carniverous or omniverous.

During the first two months, the first two infants were given four meals a day, and the last infant, only three meals a day; it was considered probable that the three meal plan was preferable.

Ordinary hospital dishes were used to serve each article, even salt being served separately; the liquids were placed in ordinary hospital tumblers. A sheet was placed underneath the table, and also for the first two or three meals one was placed on top of the table and pinned around the infant's neck at the rear in order to collect any fragments that fell.

The beef was served raw and cooked; all other meats were served cooked. The bone marrow was served cooked and raw. The vegetables were finally divided, but not sifted; carrots, lettuce, cabbage and peas were served both raw and cooked.

The cereals were all whole cereals.

Apples were served cooked and raw, the other fruits raw, but finely divided.

The nurse was instructed to sit quietly by the infant and not to make a motion until the infant did something himself. If the infant reached for food, she picked up a teaspoonful, and if he opened his mouth, she put it in, but she was not permitted to attract his attention from any food in any way or to refuse him any food for which he asked.

Two of the infants were not interested in being fed by the nurse, and first tried to feed themselves by putting their faces down into the dish; they soon learned that this was not practical, and adopted the method of eating foods with their hands.

It was found utterly impossible to predict what the infants would do at any one meal, and it was learned that there was no regularity as far as quantity of the individual ingestion of food was concerned. For instance, one infant ate four bananas, and the smaller one had 900 Gm. of orange sections at one time. The amounts of milk varied from 11 to 48 ounces (325 to 1,419 cc.) a day.

The weights have all been above the average for the age and height, but the infants are not fat.

QUESTION: What is the cost per calory of this type of feeding?

DR. DAVIS: I have never had the courage to ask the hospital management what the expense of this has been. I feel that it has been rather grave.

QUESTION: How long does a meal take?

DR. DAVIS: On an average, twenty minutes. Usually at the end of twenty minutes; the child has eaten steadily but without hurry during the entire time, and he will stop. Usually after a few weeks he will begin to offer the nurse something to eat, having satisfied himself, and he may take a few bites more, but is practically through after twenty minutes.

Any time that he completes the amount of food furnished, more is provided the next time it is served, and, of course, not every article can be served each time. The feeding is divided into four meals.

QUESTION: After they get accustomed to it, do they play with the food or throw much away?

DR. DAVIS: It is surprising how neat the eating becomes after a while. The older child spills practically nothing. The bib has been weighed, and it weighs practically the same. The bib of the smaller child weighs an ounce more after the feeding, this being largely liquid — water, orange juice or milk — that is spilled.

QUESTION: How are the eggs served?

DR. DAVIS: Both cooked and raw.

# Book Reviews

POLIOMYELITIS. W. RUSSEL MACAUSLAND. Price, $5.50. Pp. 386, with 173 engravings. Philadelphia: Lea & Febiger, 1927.

This is a well written and complete exposition of the present day status of the subject of infantile paralysis, especially from the standpoint of treatment. It is a modern, comprehensive account of the disease, one that presents practically all its phases, especially the principles of treatment.

It is the clearest and most concise exposition of this important subject that has been presented in one book. It exposes a wider knowledge of the clinical manifestations, a clearer understanding of the pathologic processes and more accurate information regarding the specific nature of the disease.

Great progress has been made in the methods of treatment and particularly in the orthopedic field. Deformity can now be prevented in nearly every instance. The return of function can be hastened by proper treatment of the involved area. And finally by the use of surgical procedures the wearing of cumbersome apparatus may be obviated in the majority of cases. From the large number of operative procedures that have been devised for the relief of paralytic conditions, the author has selected those measures that have stood the test of time and that can be accepted as standard.

Chapters on epidemiology, etiology, pathology, symptomatology and the classification of the types of poliomyelitis are well written. The diagnosis in the preparalytic and the paralytic stages is given. Prognosis in general is given and the treatment in detail. A short chapter on apparatus and on plaster of paris and the technic of its use is of value. The subject of paralysis of the trunk, including paralytic scoliosis, kyphosis, lordosis and abdominal paralysis is treated. The value of heliotherapy, massage, heat, hydrotherapy, electricity and muscle reeducation is discussed in considerable detail. Practically every operative procedure that has been proved of value is discussed. The illustrations are excellent. A complete bibliography accompanies each chapter.

The author emphasizes the great importance of the prevention of the development of deformities and the necessity of their early correction when they have occurred. When the value of these principles is impressed on the minds of all surgeons, the number of deformities in infantile paralysis will be almost negligible. The possibility of the restoration of function to paralyzed limbs is greater today than ever before due to the therapeutic and operative measures at one's disposal.

The author believes that it is feasible that a serum test might be worked out and made on children of the most susceptible age to determine the possibility of establishing individual susceptibility. He believes that there is little doubt that human carriers play a part in the diffusion of poliomyelitis since the virus has been proved to exist on the mucous surfaces. He states that in some instances evidence has pointed to foodstuffs as the carriers of the infection. Concerning the various types of serums, he states that at the present time it is impossible to judge the efficacy of any kind of serum whether normal or immune, animal or human.

The prevention of deformity and the correction of deformity as soon as it is recognized are the most important factors in the treatment of infantile paralysis. Since we have a complete knowledge of the etiologic factors that enter into the formation of deformity, and have means at our disposal to prevent and correct malposition, few cases of structural deformity should ever develop. One must be on the alert for the occurrence of deformity at all times. As soon as recog-

nized, at whatever stage of paralysis, it should be corrected. Mechanical means, manipulative measures and operative treatment should be used as indicated.

This book should be of great value to the general practitioner who is the one who usually sees these cases first and whose judgment determines, to a marked degree, the ultimate result of recovery of function. It is a valuable book to the pediatrician, to the neurologist and to the orthopedic surgeon.

AVITAMINOSEN UND VERWANDTE KRANKHEITSZUSTAENDE. By W. STEPP and P. GYOERGY. Price, 66 R. M. Pp. 817. Berlin: Julius Springer, 1927.

This is a timely work on a subject which has been developed during the past ten years. It differs from other books on this subject in maintaining the clinical rather than the laboratory point of view. One of the authors is a clinician and at the same time was one of the first to study the deficiency diseases experimentally; the other is a children's specialist, who during the past few years has carried out a large series of investigations in the clinic on both rickets and tetany.

The first chapter is on the experimental basis of vitamin studies by Stepp. The next considers the pathologic anatomy of the experimental avitaminoses, and has been well covered by Kihn. Several other writers have collaborated with the main authors in the preparation of various subjects; for example, adult scurvy has been contributed by Salle, beriberi by Shimazono of Tokyo, ship beriberi by Nocht of Hamburg, pellagra by Lavinder of New York, the edema disease by Schittenhelm of Kiel, and sprue by Walther Fischer. In addition to these subjects, xerophthalmia, rickets, infantile tetany, osteomalacia and "idiopathic" tetany of adults, infantile scurvy and alimentary anemia in infants and older children are considered by Stepp and Gyoergy.

Although one may differ from some of the points of view, as is natural in a subject of this kind which is at present undergoing such rapid changes, on the whole the treatment of the various subjects is well founded on experimental and clinical observations and considered in the light of the most recent literature. The book is illustrated with numerous charts and photographs and contains adequate bibliography at the end of each chapter. It can be highly recommended for all who wish to get a comprehensive picture of the status of the vitamins at the present time.

DER KRAFTWECHSEL DES KINDES. By DR. EGON HELMREICH, Assistant in the University Children's Clinic in Vienna, with an introduction by Dr. Clemens Pirquet, Director of the University Children's Clinic in Vienna. Price, 6 marks. Pp. 113, with 21 figures. Vienna: Julius Springer, 1927.

This small handbook is intended to give to the practicing physician the essential facts concerning energy metabolism in infants and children. The significance of the heat exchange of the body is discussed with especial reference to the characteristics of the metabolism in young children. The influences of age, body weight and build, state of nutrition, temperature, food intake and activity on the metabolism are considered. The alterations in the metabolism occurring as the result of such pathologic conditions as the infectious diseases, endocrine disturbances, obesity, and diseases of the blood are mentioned. Brief descriptions of the spirometers of Krogh and of Helmreich and Wagner and the respiration apparatus of Benedict are given. One chapter is devoted to case reports illustrating the characteristic changes in the energy metabolism occurring as the result of pathologic conditions. A bibliography of the more important contributions to the study of energy metabolism in children is appended. The book as a whole contains little that is new, but it is, nevertheless, a good resumé of the subject.

PATHOLOGICAL PHYSIOLOGY OF INTERNAL DISEASES. ALBION W. HEWLETT, M.D., B.S. Third edition. Revised in Memoriam by His Colleagues. Pp. 787, with 164 illustrations. New York: D. Appleton & Company, 1928.

The third edition of this standard work has been carefully revised and well written. The original form and order of presentation have been largely retained, and many of the recent significant advances in clinical physiology have been included.

This book deals with those changes in function that are presented by patients suffering from internal diseases. It is an attempt to combine the science of medicine with the art of medicine. By this method the reader finds physiology, pathology and symptoms, all explaining the existing clinical conditions.

The same criticism may be applied to this book that is true of all others of this type. The subject matter in general is too detailed for the general reader, while for reference to any particular subject, it is too brief. At the end of each chapter, however, there is a good list of references, and monographs on particular subjects.

AUTOBIOGRAPHY OF OTTO HEUBNER. Price, R. M. 12.60. Pp. 210. Berlin: W. Heubner, 1927.

The autobiography of Otto Heubner is of unusual interest to the pediatrician because it tells the transition period of German pediatrics, when it developed from a much neglected specialty to a full fledged university department on a par with medicine, surgery and obstetrics.

Trained in pathology and internal medicine, Professor Heubner came in the field of pediatrics splendidly equipped, and his thorough clinical work is a monument to this training. His personal experiences as a defender of the use of diphtheria antitoxin are of interest and the added difficulties in its introduction following the death of Professor Langerhans' son. The book is well worth the reading by all interested in the historical development of German pediatrics.

UNIVERSITY OF IOWA STUDIES. Volume 3. Price, $1. Iowa City, Ia.: University of Iowa.

This is the collection of forty articles published by the members of the medical faculty of the University of Iowa, each paper being presented in reprint form. There is a wide range of subjects covered.

# Directory of Pediatric Societies

## NATIONAL

### AMERICAN MEDICAL ASSOCIATION, SECTION OF DISEASES OF CHILDREN

Chairman: Dr. Hugh McCulloch, 500 S. Kingshighway, St. Louis.
Secretary: Dr. C. A. Aldrich, 545 Lincoln Ave., Winnetka, Ill.
Place: Minneapolis. Time: June 11-15, 1928.

### AMERICAN PEDIATRIC SOCIETY

President: Dr. J. Claxton Gittings, 1828 Pine St., Philadelphia.
Secretary: Dr. Howard Childs Carpenter, 1805 Spruce St., Philadelphia.
Place: Washington, D. C. Time: April 30 to May 2, 1928.

### THE ASSOCIATION OF AMERICAN TEACHERS OF THE DISEASES OF CHILDREN

President: Dr. J. P. Crozier Griffith, 1810 Spruce St., Philadelphia.
Secretary: Dr. Paul W. Emerson, 86 Bay State Road, Boston.
Place: Minneapolis. Time: June 12, 1928.

### AMERICAN CHILD HEALTH ASSOCIATION

President: Hon. Herbert Hoover, 370 Seventh Ave., New York.
Secretary: Dr. Philip Van Ingen, 370 Seventh Ave., New York.
Place: Stevens Hotel, Chicago. Time: October 15-19, 1928.

### CANADIAN SOCIETY FOR THE STUDY OF DISEASES OF CHILDREN

President: Dr. G. R. Pirie, 20 Madison Ave., Toronto.
Secretary: Dr. A. P. Hart, 1844 Bloor St. West, Toronto.
Place: Vancouver, B. C. Time: June, 1928.

## SECTIONAL

### CENTRAL STATES PEDIATRIC SOCIETY

President: Dr. H. T. Price, 121 University Place, Pittsburgh.
Secretary: Dr. A. H. Parmelee, 310 S. Michigan Ave., Chicago.

### SECTION ON PEDIATRICS OF SOUTHERN MEDICAL ASSOCIATION

Chairman: Dr. E. C. Mitchell, 1073 Madison Ave., Memphis, Tenn.
Secretary: Dr. A. J. Waring, De Renne Apts., Savannah, Ga.
Place: Asheville, N. C. Time: November 12-15, 1928.

## LOCAL

### NORTHERN CALIFORNIA PEDIATRIC SOCIETY

President: Dr. W. L. Channell, Medical Bldg., Oakland, Calif.
Secretary: J. E. Parrott, Medico-Dental Bldg., San Francisco.
Place: Various Teaching Schools. Time: Monthly.

### SOUTHWESTERN PEDIATRIC SOCIETY

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary and Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: University Club of Los Angeles. Time: First Wednesday in January, March, May, September and November.

### THE ROCKY MOUNTAIN PEDIATRIC SOCIETY

President: Dr. W. Wiley Jones, Majestic Bldg., Denver.
Secretary: Dr. Berryman Green, 537 Republic Bldg., Denver.
Place: Children's Hospital, Denver. Time: Third Saturday of each month, 2:30 p. m., September to May, inclusive.

### GEORGIA PEDIATRIC SOCIETY

President: Dr. Joseph Yampolsky, 127 Peachtree St., Atlanta.
Secretary: Nevin M. Atkins, Atlanta.
Place: Savannah, Ga. Time: May 9, 1928.

### PEDIATRIC SECTION OF FULTON COUNTY MEDICAL SOCIETY, ATLANTA, GA.

President: Dr. William W. Anderson, 362 Peachtree St., Atlanta.
Secretary: Dr. Lee Bivings, Exchange Bldg., Atlanta.
Place: Academy of Medicine, 32 Howard St., Atlanta.
Time: Second Thursday of each month from October to April, at 8 p. m.

### NEW ENGLAND PEDIATRIC SOCIETY

President: Dr. James Herbert Young, 66 Commonwealth Ave., Boston.
Secretary: Dr. Thomas Lanman, 286 Marlborough St., Boston.
Place: Boston Medical Library. Time: Second Friday of each month.

### CHICAGO PEDIATRIC SOCIETY

President: Dr. Maurice Blatt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. J. K. Calvin, 104 S. Michigan Ave., Chicago.
Place: City Club. Time: Third Tuesday of each month.

### LOUISIANA STATE PEDIATRIC SOCIETY

President: Dr. L. R. De Buys, 1487 Delachaise St., New Orleans.
Secretary: Dr. F. J. Kinberger, 3601 Prytania St., New Orleans.

### DETROIT PEDIATRIC SOCIETY

President: Dr. George E. White, Capitol Theatre Bldg., Windsor, Ontario, Canada.
Secretary: Dr. W. S. O'Donnell, 1563 David Whitney Bldg., Detroit.
Place: Children's Hospital of Michigan. Time: No fixed date; approximately once a month.

## NORTHWESTERN PEDIATRIC SOCIETY

President: Dr. F. W. Schlutz, University of Minnesota, Minneapolis.
Secretary: Dr. E. S. Platou, 730 La Salle Bldg., Minneapolis.
Place: University Campus and Rochester. Time: Winter, Spring, Summer, Fall.

## ST. LOUIS PEDIATRIC SOCIETY

President: Dr. Park J. White, Beaumont Bldg., St. Louis.
Secretary and Treasurer: Dr. Ralph L. Cook, 508 N. Grand Blvd., St. Louis.
Place: St. Louis Children's Hospital. Time: At least once a year and contingent thereafter on the arrangement of a suitable program.

## NEBRASKA-IOWA PEDIATRIC SOCIETY

President: Dr. Floyd Clarke, 731 City Nat'l Bank Bldg., Omaha.
Secretary: Dr. R. B. Eldredge, 1036 Medical Arts Bldg., Omaha.
Place: Add-Sell Restaurant. Time: Second Thursday of each month from October to May, inclusive, 6 p. m.

## BROOKLYN PEDIATRIC SOCIETY

President: Dr. Louis M. Ruderman, 961 Eastern Parkway, Brooklyn.
Secretary: Dr. Joseph F. Paulonis, 1357 Bushwick Ave., Brooklyn.
Place: Kings County Medical Society Bldg. Time: Last Wednesday of each month, except June, July and August.

## BRONX PEDIATRIC SOCIETY

President: Dr. William Hinz, 1964 Grand Concourse, New York.
Secretary: L. H. Barenberg, 1749 Grand Concourse, New York.
Place: Concourse Plaza, 161st, St., and Grand Concourse, New York.
Time: Second Wednesday of each month, except June, July, August and September.

## CENTRAL NEW YORK PEDIATRIC CLUB

President: Dr. Frank Vander Bogert, 111 Union St., Schenectady, N. Y.
Secretary: Dr. Frank J. Williams, 58 S. Swan St., Albany, N. Y.
Place: Various Cities in New York. Time: Fourth Thursday of October, January and April.

## PEDIATRIC SECTION OF THE NEW YORK ACADEMY OF MEDICINE

President: Dr. William Ropes May, 8 East 66th St., New York.
Secretary: Dr. Stafford McLean, 17 East 71st St., New York.
Place: New York Academy of Medicine, 2 East 103d St. Time: Second Thursday of each month from October to May, inclusive, 8:30 p. m.

## THE PEDIATRIC SECTION OF THE ACADEMY OF MEDICINE OF CLEVELAND

Chairman: Dr. J. W. Epstein, 3912 Prospect Ave., Cleveland.
Secretary: Dr. Charles W. Burhans, 14805 Detroit Ave., Lakewood, O.
Place: 2009 Adelbert Road. Time: Four meetings, from September to May.

## PEDIATRIC SECTION, ACADEMY OF MEDICINE, TORONTO

President: Dr. M. A. Cox, 34 St. Clair Ave. West, Toronto.
Secretary: Dr. F. R. Hassard, 1361 King St. West, Toronto.
Time: November 17, December 15, February 16 and March 15.

## PHILADELPHIA PEDIATRIC SOCIETY

President: Dr. Edward L. Bauer, Jefferson Medical College, Philadelphia.
Secretary: Dr. John Porter Scott, 2040 Pine St., Philadelphia.
Place: College of Physicians, 19 S. 22nd St., Philadelphia. Time: Second Tuesday of each month from October to June, inclusive.

## MEMPHIS PEDIATRIC SOCIETY

President: Dr. W. D. Blue, 1024 Madison Ave., Memphis, Tenn.
Secretary: Dr. Gilbert J. Levy, 899 Madison Ave., Memphis, Tenn.
Place: Memphis General Hospital. Time: Second Tuesday of each month.

## TEXAS PEDIATRIC SOCIETY

President: Dr. Lucius D. Hill, Medical Arts Bldg., San Antonio.
Secretary and Treasurer: Dr. Edwin G. Schwarz, Medical Arts Bldg., Fort Worth.
Place: Galveston. Time: May 9, 1928.

## RICHMOND PEDIATRIC SOCIETY

President: Dr. Howard Urbach, 1331 Grove Ave., Richmond, Va.
Secretary: Dr. W. Ambrose McGee, West End Medical Bldg., 2618 Grove Ave., Richmond, Va.
Place: Westmoreland Club, 601 E. Grace St., Richmond, Va.
Time: First Thursday of each month (other than during the summer).

## VIRGINIA PEDIATRIC SOCIETY

President: Dr. Lawrence T. Royster, University, Va.
Secretary: Dr. William B. McIlwaine, Petersburg, Pa.
Place: Danville, Va. Time: October 17, 1928.

## NORTH PACIFIC PEDIATRIC SOCIETY

President: Dr. R. S. Miles, Fidelity Bldg., Tacoma, Wash.
Secretary: Dr. D. M. Dayton, 704 St. Helens Ave., Tacoma, Wash.

## SEATTLE PEDIATRIC SOCIETY

President: Dr. George B. McCulloch, Cobb Bldg., Seattle.
Secretary: Dr. C. L. Smith, Stimson Bldg., Seattle.
Place: College Club. Time: Third Friday of each month, at 7 p. m.

## MILWAUKEE PEDIATRIC SOCIETY

President: Dr. A. B. Schwartz, 418 E. North Ave., Milwaukee.
Secretary: Dr. M. G. Peterman, 167 Seventeenth St., Milwaukee.
Place: Children's Hospital, Milwaukee. Time: Second Wednesday of each alternate month, beginning with February.